The Australian and New Zealand Journal of Surgery

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VOLUME I—1931-1932

SYDNEY
AUSTRALASIAN MEDICAL PUBLISHING COMPANY, LIMITED

UNIV OF CALIF
MEDICAL SCHOOL

The Australian and New Zealand Journal of Surgery

JUNE, 1931.

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THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

Registered at the General Post Office, Melbourne, for transmission by post as a periodical.

VOL. I. JUNE, 1931. No. 1.

PRESIDENT'S ADDRESS.[1]

By SIR HENRY NEWLAND.

My first very pleasant duty will be to convey to your, Sir, in the name of the assembled Fellows of this College, our thanks for the honour that you have done us in coming here this evening to open the Fourth Annual Meeting of the College. We feel that the doings of medicine men outside of the sick room have usually but a doubtful interest for those whose energies are expended in channels other than those that belong to the art of healing; nevertheless, we are assured, by the kindly and understanding address you have just delivered, that our profession and our aims are by no means without interest for you.

During the year that has passed since our last annual meeting in Melbourne, the affairs of the College have provided the scene of much activity; and the first announcement I have to make, one that is at once gratifying and appropriate, is that His Majesty the King has been graciously pleased to approve of the prefix "Royal" to the title of the College.

We feel that this great honour possesses imperial significance; and that our proposed new title, "The Royal Australasian College of Surgeons," will proclaim that the surgeons of Australia and New Zealand will cherish and maintain British traditions, and uphold the ideals of the parent College of England.

The College has been further honoured by His Majesty by a grant of arms through the College of Heralds; and, as a mark of special favour, supporters for the arms have been granted.

A further distinction which has occasioned the warmest feelings of gratitude and pleasure, has been the announcement of the projected gift of a mace; this is to be the gift of the President and members of the

[1] Delivered at the Fourth Annual Meeting of the College of Surgeons of Australasia, Sydney, March, 1931.

Council of the Royal College of Surgeons of England. The mace is to be a replica of the mace borne before the President of the Royal College on state occasions, except that it will have the arms of the two Colleges and of the Commonwealth and Dominion. His Majesty has graciously given permission for the Orb and Cross to be included in the design.

If anything could heighten our sense of obligation to the Council of the Royal College of Surgeons of England for the generous gift, it would be the announcement that the President, Lord Moynihan, hopes to make the long journey to present the mace in person at the next annual meeting of this College in 1932.

Another striking activity of the College arose out of the suggestion that the Primary Examination for the F.R.C.S. diploma of the English College should be conducted here by special examiners sent out for the purpose by the English College for the benefit of Commonwealth and Dominion students and practitioners.

A vast amount of work and correspondence has been expended on this subject; special classes for candidates have been organized in Sydney and Melbourne and a sufficient number of aspirants have announced their candidature. The final result of this attempt to render the coveted diploma a little more accessible remains to be seen.

During the past year, the College has achieved a corporate existence, by incorporation under the *Companies Act* of the State of Victoria. This enables the College to hold property, to receive bequests, and confers many other advantages. It is felt, however, that this incorporation should be replaced by incorporation under Royal Charter, as the College is an institution which embraces two great Dominions of the Empire. An Imperial Charter will add to the dignity and authority of the College, and will permit registration of its diploma. Now that the College has a corporate existence, the way has been opened for the acquisition of land on which to erect a permanent headquarters, and Fellows will be asked to decide upon a site for a building to house the administrative offices of the College, and a museum and library.

The two most important duties of the College are the training of surgeons and the promotion of research in surgery. In admitting a candidate to the Fellowship, the College takes into consideration three factors, namely, that his personal character shall be above reproach, that he shall have a sound knowledge of the basic principles of surgery, and that he shall have served a period of surgical apprenticeship as assistant in a recognized surgical clinic. In order to insure that he shall have been well grounded in the principles of surgery, the possession of a senior surgical qualification will be demanded after February of next year. In relation to this matter, it gives me much pleasure to report that the regulations, governing admission to the M.S. degree of the three Australian Universities, have now been made practically uniform, an achievement that has been rendered possible largely by means of the work done by this College. The College will encourage all applicants for its Fellowship to sit for a senior degree in surgery

in their own Australian Universities. It will also demand from them a period of apprenticeship in surgery. It does not necessarily follow that because a man possesses a senior surgical degree, he is a good surgeon. He must, in addition, possess technical skill, which can only be obtained by assisting a master of his craft. The College proposes to appeal to all large general hospitals to provide facilities for the training of the future surgeons of Australasia. It does not believe that a period of a few months spent as a house surgeon is an adequate surgical training, though it is possible, during this time, to equip a graduate with sufficient knowledge to cope with the ordinary surgical emergencies. In order to become a good surgeon, some years must be spent as an assistant and it is believed that this can be arranged at the hospitals without interfering with the training of future general practitioners. The College demands a period of five years' training after graduation from applicants for Fellowship, and, at the end of this period, a Board of Censors in each State and in the Dominion will personally satisfy itself regarding the surgical judgement and ability of each candidate. I think this procedure will insure that possession of the Fellowship will be evidence that a candidate has undergone the best type of surgical training.

The other important function of the College is the promotion of surgical research; and our work, in this respect, has received a great stimulus from the generous gift of £2,500 by Lady Syme and her children in memory of the late Sir George Syme, our first President. The interest from this sum is to be devoted to the foundation of an annual oration and a research scholarship, which will both be named after our late President who did so much to aid in the foundation of this College. It is proposed to encourage research in surgical diseases common in Australia; and, in regard to this, I wish briefly to refer to the Registry of Hydatid Disease in Australia and New Zealand, which has been begun, during the year, at the instigation of the Vice-President, Sir Louis Barnett, and New Zealand Fellows. I appeal to all Fellows to aid in this work, for the knowledge gained by an analysis of these cases will be of great help in advancing the treatment of this disease. This is a work which can be undertaken only by such a representative body as this College.

This somewhat rapid survey of the activities of the College during the past twelve months would not be even approximately complete without an allusion to the services rendered by H. B. Devine, who converted a large portion of a much needed holiday in Europe into a time of hard work in the interests of the College with conspicuous ability and success.

THE TECHNIQUE OF WERTHEIM'S OPERATION.

By VICTOR BONNEY,

Gynæcological Surgeon to Middlesex Hospital; Surgeon to Chelsea Hospital for Women et cetera.

Of all standard operations designed for the extirpation of cancer, Wertheim's operation for carcinoma of the cervix has the largest percentage of permanent cures. Up to the end of 1930 I had performed it four hundred and seven times. The operative mortality for the whole series is 14%, but for the last hundred cases 6%. Up to the end of 1925 I had performed it three hundred and ten times, with the following results:

Died of the operation	51
Lost sight of before five years	11
Died other disease before five years	8
Died recurrence before five years	116
Well after five years	124*
	310

* 40% of patients operated on.

These results are not exceptional, but agree with those published by all surgeons who have practised the operation on a large scale. As, however, the object of this paper is to describe the technique of the operation, I will refer those interested in the results to two former communications of mine in which comparison is made between them and the best results obtained by radium.[1] [2]

Wertheim's operation has been decried on account of its operative mortality and its difficulty. The mortality at its beginning was high, for Wertheim lost 30 patients out of his first 100, whilst my mortality for my first 100 cases was 20%. Increased experience and improvement in operative technique has, however, enabled me to bring it down to 6% for my last 100 cases.

As regards the difficulty of the operation, there has been much exaggeration. It is difficult, of course, but not beyond the powers of any good surgeon, provided he will take the trouble to learn the technique and has the opportunity of performing it sufficiently often. The correct technique of any operation is best acquired by assisting one who is already a master of it, but a great deal may be learned from written description. Now, it is a curious fact that whereas every other regular major operation is minutely described in the text books, Wertheim's operation lacks any adequate description, excepting that published originally by its inventor, and that to be found in the book on gynæcologi-

cal surgery written by Comyns Berkeley and myself. The opinion I have formed from reading many gynæcological text books is that the authors, though purporting to describe the operation, have never really performed it. The illustrations I have drawn to show the successive steps of the operation should be studied with the text, but the reader must bear in mind that if a drawing of a pelvic operation is to show anything at all it is necessary for the artist to "superficialize" the parts concerned, and thus make the procedure appear more easy than it is.

TECHNIQUE OF THE OPERATION.

Operable Cases.—A case is operable if: (i) the bladder is not involved, (ii) the rectum is not involved, (iii) both ureters are not involved, (iv) the malignant mass or its secondary extensions are not firmly adherent to the pelvic side wall or the great vessels running along it. Involvement of the bladder produces urinary changes and painful and frequent micturition. If these are absent, the bladder is free. Involvement of the rectum can be felt. Involvement of both ureters can only be guessed at. It does not happen till quite late in the disease and the patient is very ill. The only way to be sure is to inspect the ureters through an abdominal incision. Fixation of the growth or its secondaries to the pelvic side wall again can, as a rule, only be guessed at, for great induration of the paracervical tissues may be merely inflammatory. As a result of my own experience I have come to the conclusion that it is impossible, short of opening the abdomen, to be certain whether an advanced growth is or is not removable. I have opened patients in the certain belief that the case would be easily operable, and found the growth irremovable; whilst in others an operation entered upon with the slenderest hope turned out to be perfectly easy. Speaking generally, if micturition is normal and the patient is in reasonable condition, the patient is worth operating on.

Previous Preparation.—Previous preparation is that of an ordinary abdominal operation. I do nothing to the growth unless it is fungating and very large, in which event it makes the operation easier if a good deal of it be cut away a week beforehand.

Anæsthesia.—I use spinal anæsthesia with full ether anæsthesia. The ether counteracts the effect that spinal anæsthesia sometimes has on feeble patients with low blood pressure. The ether anæsthesia should be full, the spinal anæsthetic being used merely to block shock impulses. The fatalities I know of with these combined anæsthesias have all been in cases in which light inhalation anæsthesia was given. Eight minutes should elapse after the spinal injection before putting the patient in the Trendelenburg posture, the patient's legs being kept flexed meanwhile and the shoulders well raised.

Packing the Vagina.—Directly after the spinal injection the catheter should be passed and the vagina packed very tightly with gauze soaked in violet-green solution. The solution antisepticizes the vagina, whilst

the gauze makes it into a solid column which greatly facilitates the subsequent separation of the bladder and rectum from it.

Position.—The Trendelenburg position is used, but with not more tilt than is necessary.

The Abdominal Incision.—The incision is ordinarily made from umbilicus to pubis in the mid-line, but in fat patients some extension above the umbilicus is advantageous. The skin edges should be covered by sheet rubber or towels.

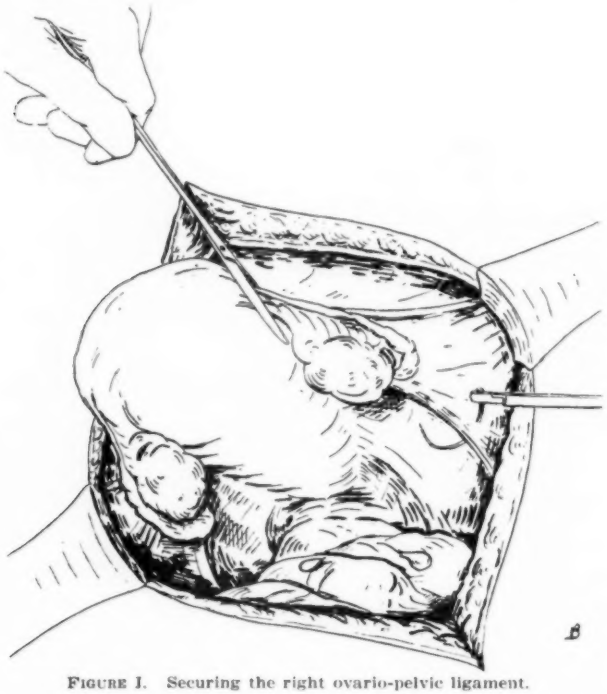

FIGURE I. Securing the right ovario-pelvic ligament.

Packing Off the Intestines.—For packing off the intestines I use several swabs about five inches square, made of wool and gauze and wrung out of warm saline solution.

Retractor.—A self-retaining retractor should now be inserted. I use Berkeley's pattern with sheet rubber attached to the blades so as to cover the wound edges. Time should be taken in packing off the intestines and inserting the retractor, as a perfect exposure of the pelvic contents is absolutely necessary, and if the patient is straining under the anæsthetic, the surgeon must wait till she is fully relaxed.

Preliminary Inspection.—The surgeon should inspect and feel the parts to find out what sort of problem he is up against. If the ureters are dilated, they can be felt and probably seen on the pelvic side wall. Infiltration of the bladder is suggested if a deep puckering of the peritoneum at the bottom of the utero-vesical pouch is seen, though this puckering may be above the bladder and thus not involve it. The

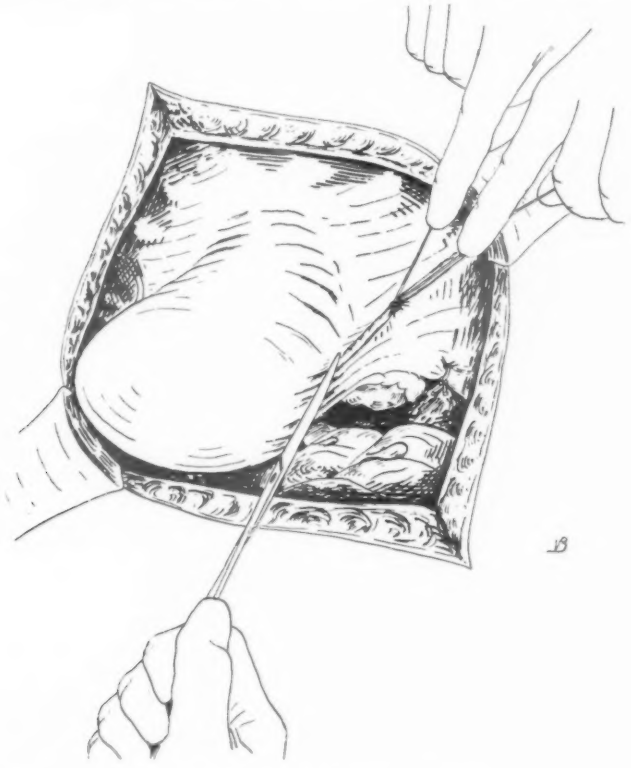

FIGURE II. Tying the round ligament.

glands along the external iliac vessels should be felt. If obviously enlarged their mobility should be tested. Malignant glands in the obturator fossæ may be felt if very large, but generally the state of these has to be ascertained later on in the operation.

Tying the Ovario-Pelvic Ligaments.—The ovario-pelvic ligaments are now under-run with a needle and ligatured as far out on the pelvic side wall as possible (Figure I).

Tying the Round Ligaments.—The round ligaments are similarly tied well outside the uterus (Figure II).

Dividing the Broad Ligaments.—A pair of long Spencer Wells forceps have been made to clamp the ovario-uterine ligament, the tube and the broad ligament, close up to the uterus, the broad ligament is divided just inside the ligatures on the ovario-pelvic and round ligaments,

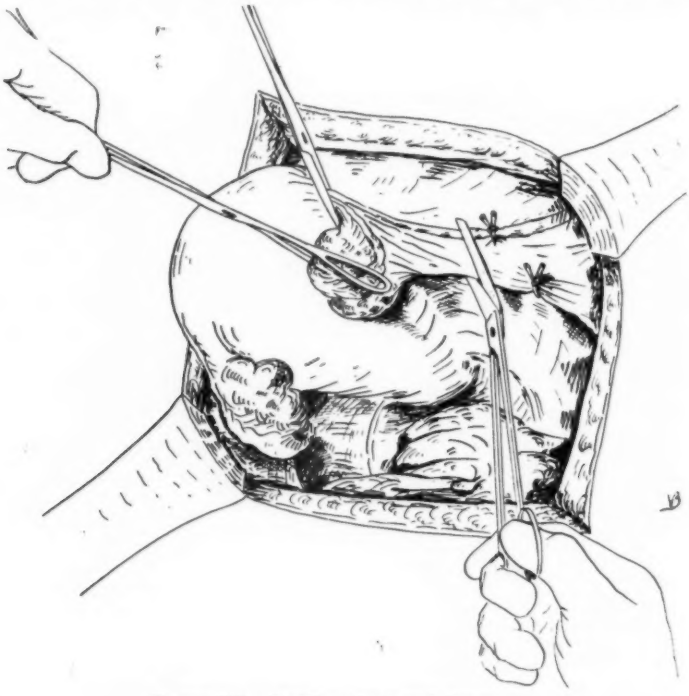

FIGURE III. Dividing the broad ligament.

and the whole appendage is freed and brought up (Figure III). The same is then done on the opposite side.

Ligaturing the Uterine Cornua.—A piece of number 6 silk is now made to take the place of the forceps on one cornu, thus controlling the uterine end of the ovarian artery. Having tied the ligature, pull the appendage across the uterine fundus and tie again. This "bunches up" the appendage and keeps it out of the way. The ends of the ligature should be left long (Figure IV). The same is now done on the opposite side, and the long ends of the two ligatures being gripped by a pair of forceps a tractor on the uterus is obtained.

Beginning the Separation of the Bladder.—The peritoneum is now divided across the front of the uterus at the junction of the movable with the fixed portion. I separate the loose peritoneum by underrunning it with my finger, for this proceeding immediately gives the plane of cleavage between the bladder and the vagina. The peritoneal flap being reflected until the bladder is reached, the latter is now separated from the cervix and upper part of the vagina. This is best

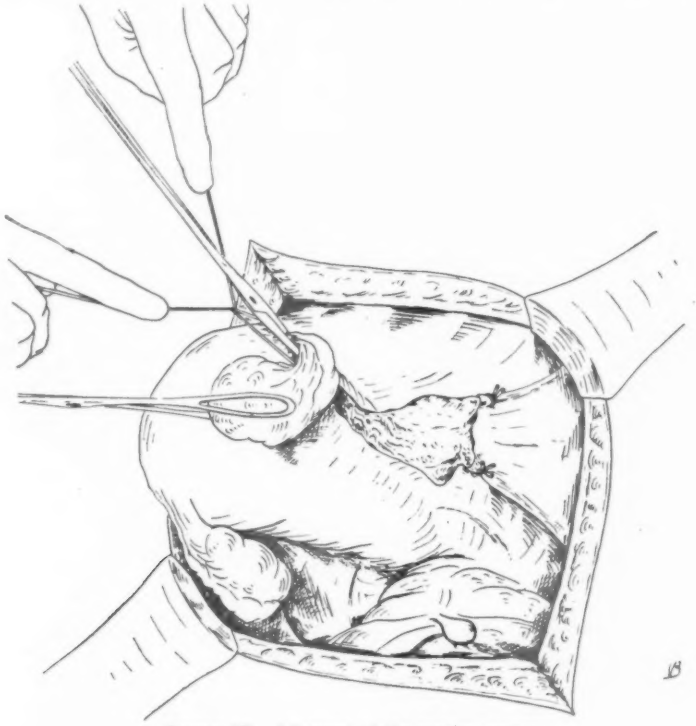

FIGURE IV. Ligature of the uterine cornu.

done by thumb pressure, the nail being kept towards the bladder whilst the pulp is pressed against the cervix and packed vagina. The correct position of the hand during this manœuvre is shown in Figure V. The thumb pressure should be supplemented by cautious snips with scissors, the points of which should be kept directed towards the vaginal wall. If the bladder is not involved, it rolls easily off the vagina, provided the proper plane of cleavage has been found; but if it is adherent by growth or inflammation, great caution is required. In such a case a broad flat retractor should be used to hold back the bladder and fully

expose its attachment to the vagina. The bladder should not at this stage be separated from more than the upper 2·5 centimetres (one inch) of the vaginal wall in the middle line and progressively less so lateral to this. The surgeon having thus assured himself that the bladder is separable, the apex of the anterior peritoneal flap should be fastened by a suture to the skin at the pubic end of the abdominal wound so as to keep the bladder out of the way.

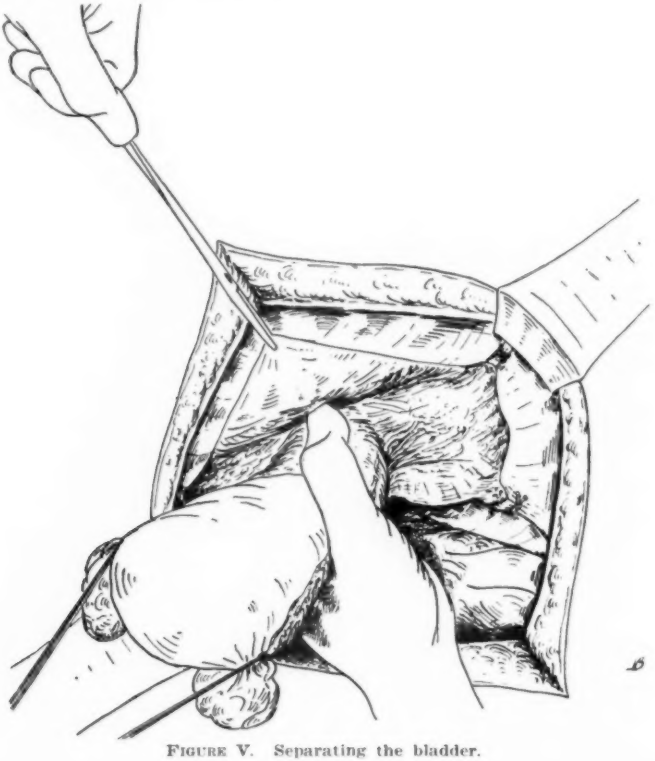

FIGURE V. Separating the bladder.

Opening up the Broad Ligament.—The broad ligament is now opened up by dividing the peritoneum between the ligatures on the stumps of the round and ovario-pelvic ligaments respectively in a line parallel to and outside the line of the ovarian vessels. Forceps are now placed on these two stumps and pulled upon, whilst with his finger and a pair of blunt-pointed scissors the surgeon teases away and divides the loose areolar tissue until he can see plainly the external iliac vein as it runs along the side wall of the pelvis.

Feeling the Ureter.—The ureter is now felt for in the manner shown in Figure VI. It is attached to the posterior peritoneal layer of the broad ligament and feels about the size of a crow quill, and it slips under the finger and thumb. The depth at which it lies varies considerably, but its feel is quite unmistakable.

Finding the Uterine Artery.—The uterus is pulled away to the opposite side, whilst the space in the broad ligament is kept open by traction on the forceps just applied. Taking the external iliac vein as his guide, the surgeon gently teases aside the tissues in the space as he searches for the obliterated hypogastric artery. This vessel runs

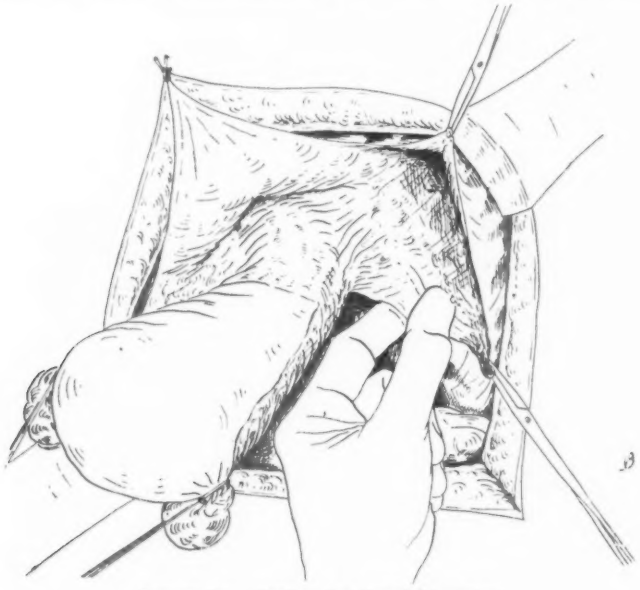

FIGURE VI. Feeling for the right ureter.

parallel to and inside the external iliac vein, but about 2·5 centimetres (an inch) deeper down, and the uterine artery normally comes off it at about a right angle. The teasing movements should be in the line of the hypogastric artery, and when it is found, the uterine artery can usually be seen coming off it (Figure VII). If it cannot be seen, the ureter should be exposed as it lies on the peritoneum, because sometimes the artery, instead of coming off the hypogastric artery at right angles, comes off at an oblique angle much nearer to the internal iliac trunk and runs towards the uterus almost parallel to the ureter, but above it. If it is not found in this situation, gently push the forefinger through the cellular tissue between the ureter and the obliterated hypogastric

artery and hook the last joint forward and upwards, when the artery will be lifted on the pulp of the finger. In advanced cases the cellular tissue may be so infiltrated that the uterine artery cannot be found by either of these devices, in which event the surgeon should follow the obliterated hypogastric artery back to the internal iliac trunk and ligature that. The big artery should be clearly seen and then lifted by seizing it

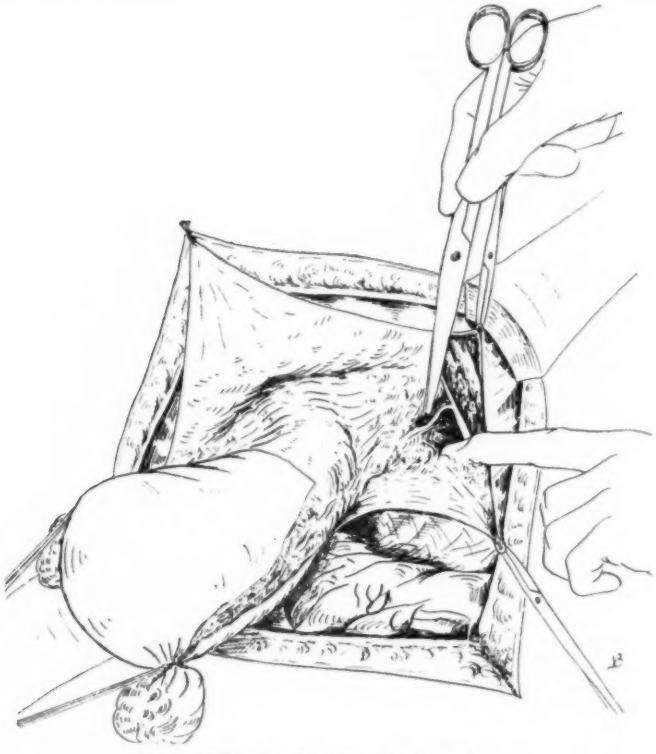

FIGURE VII. Finding the uterine artery.

with forceps, after which a stout silk ligature is placed round it by means of an aneurysm needle or, better still, by a curved blunt Reverdin needle. In the vast bulk of cases, however, the uterine artery will be found by one or other of the methods described and, when found, should be divided between forceps and the proximal end immediately tied.

Exposing the Ureter under the Uterine Artery.—By traction on the forceps holding the distal end the artery is then "rucked back" towards the uterus, thereby exposing the ureter underneath it (Figure

VIII). The distal end is now tied and the surgeon then proceeds to find the uterine artery, tie and divide it, and expose the ureter on the opposite side.

Isolating the Ureters.—The ureter having been clearly exposed at the point where the uterine artery crosses it, the entrance to the ureteric canal is felt for by following the line of the ureter downwards with the tip of the finger. The canal, which is easily entered with the finger tip if the tissues are not infiltrated, should be opened up along its roof

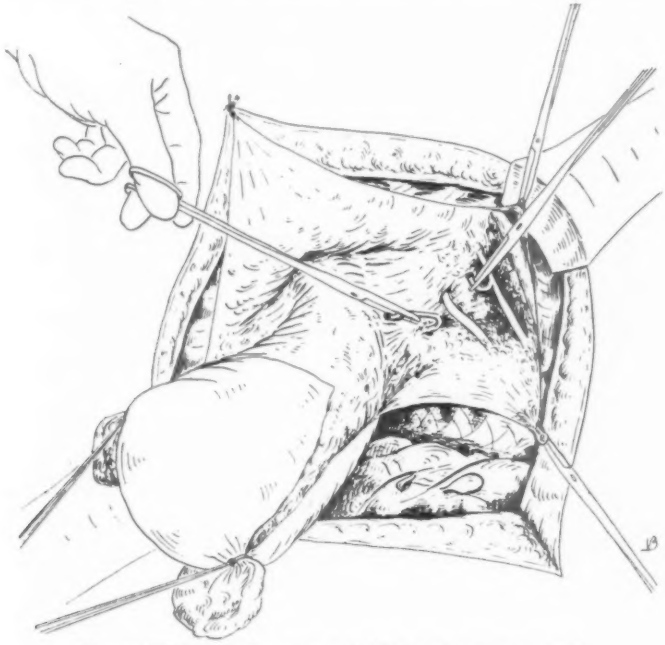

FIGURE VIII. Uterine artery divided and ureter exposed.

by a succession of scissor snips, between each of which the finger tip pushes the ureter outwards so as to avoid injuring it (Figure IX). The roof of the canal is divided until the uretero-vesical junction can be seen. By scissor snips along its inner side the ureter is now partly freed from its bed and still further pushed outwards (Figure X). A broad flat retractor holds back the bladder meanwhile, so that a clear view of the parts is obtained. The ureter on the opposite side is now treated in the same way.

Further Separation of the Bladder.—The bladder being retracted, the surgeon, by a series of scissor snips aided by thumb pressure in the

manner already described, still further separates it and the ureterovesical junctions from the vagina and the tissues lateral to it.

Separating the Vagina from the Rectum.—The uterus is now pulled right forwards and the peritoneum on the anterior wall of Douglas's pouch is cut through with the scissors just below the point where it becomes fixed on the cervix (Figure XI). Into the hole thus made the

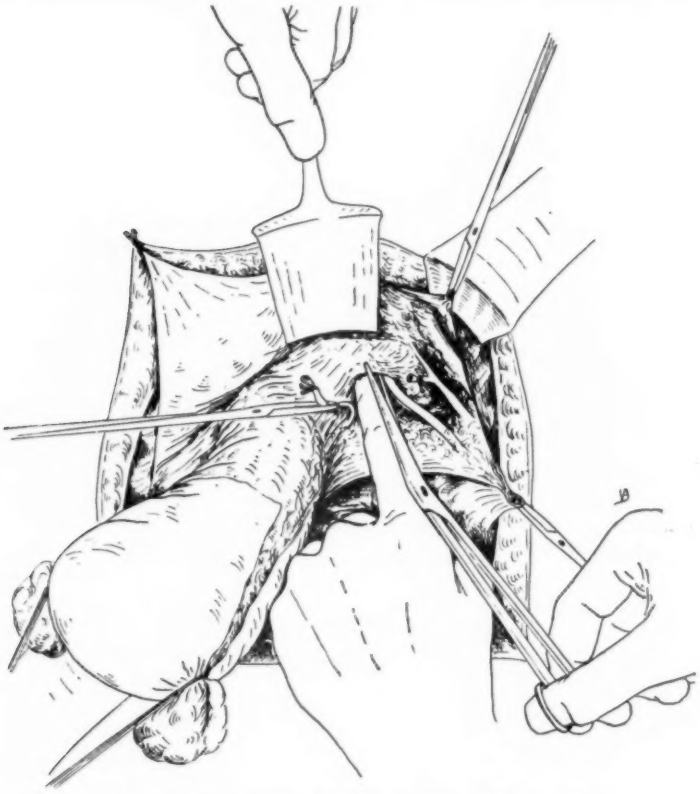

FIGURE IX. Dividing the roof of the uteretic canal.

first and second fingers of the surgeon's left hand are pushed and, keeping their pulps pressed against the solid column formed by the packed vagina, the rectum is separated off it in the mid-line (Figure XII). Where no infiltration exists, the plane of cleavage is easily found, and once it is entered on, the bowel readily becomes stripped off the vagina down to the levator floor. It is essential to keep the finger pulps

pressed hard against the packed vagina while the separation is effected by the pressure of the nails. There is now a central hole through the anterior wall of Douglas's pouch. The peritoneum lateral to this is the undivided remains of the posterior layers of the broad ligaments. The ureter having been kept out of the way by the first and second finger

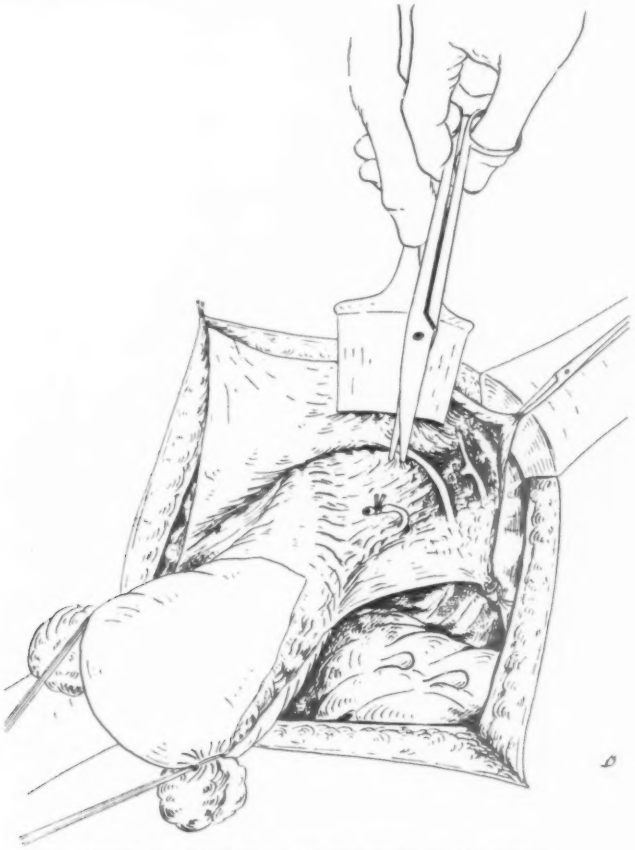

FIGURE X. Completing the separation of the ureter.

of the left hand, as shown in Figure XIII, this peritoneum is now divided down to the central hole on one side and then on the other.

Exposure of the Cardinal Ligaments.—It will now be apparent that extending outwards from the vagina are a couple of broad fan-like expansions, one on either side, which anchor the vagina to the lateral

pelvic wall and the floor of the pelvis. These are the ligaments first called "cardinal" and accurately described by the Australian surgeon Nyulasy, to the free exposure of which all the preceding steps of the operation have been working up. On their upper and anterior surfaces lie the ureters and the portions of the bladder lateral to the vagina. Their posterior and lower surfaces lie against the lateral ligaments of the rectum, but separated from them by "wodges" of fibro-fatty tissue.

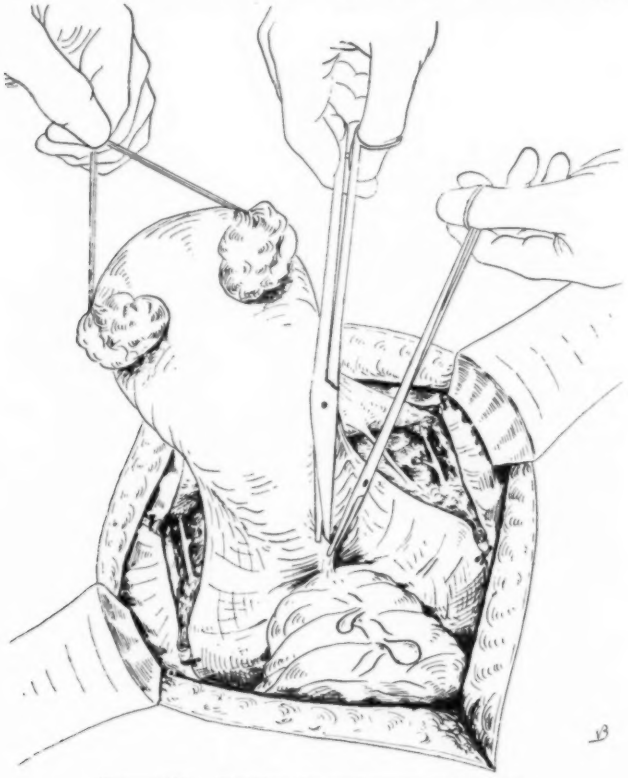

FIGURE XI. Dividing the peritoneum posteriorly.

Dividing the Cardinal Ligaments.—The forefinger is pushed in behind the ligament of one side and, guided by it, the ligament is divided by successive snips of the scissors as far outside the lateral aspect of the vagina as possible (Figure XIV). It will be found that the plane of the ligament is a curved one. It must therefore be divided successively, the surgeon between each snip making sure that the rectum is clear of it. The ligament, to begin with, should not be divided deeper

than half way down the vagina, and, this point being reached, the surgeon should turn to the ligament on the opposite side and treat it similarly. The uterus is now pulled right forwards again and the rectum inspected. The fibro-fatty masses of tissue lateral to it, already described, will need cautious dividing, whilst the rectum in the middle line is pushed back off the vagina right down to the pelvic floor, aided by occasional scissor snips (Figure XV).

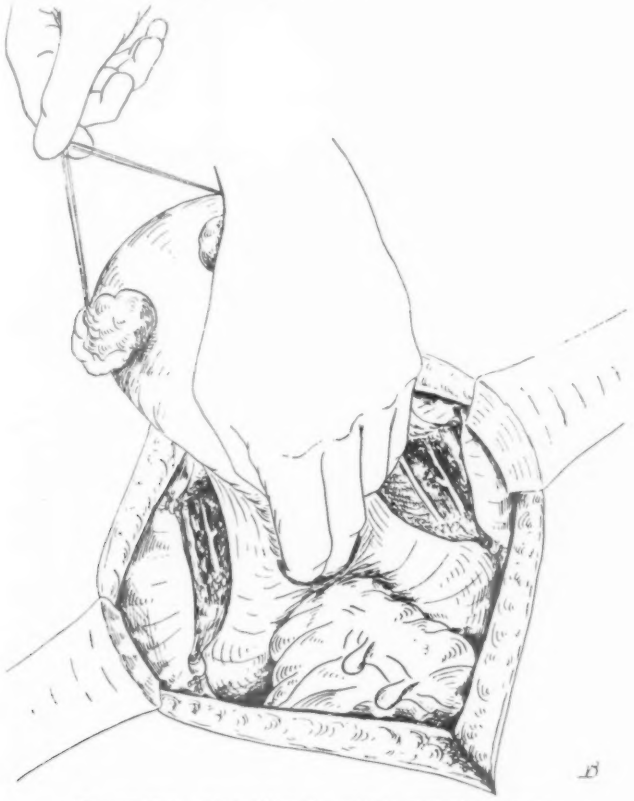

FIGURE XII. Separating the vagina from the rectum.

Completing the Division of the Cardinal Ligaments.—The bladder being held back by the flat retractor, its separation from the vagina is now carried on right down to the pelvic floor, whilst laterally the ureters are freed from the surface of the cardinal ligaments and pressed outwards. The length of ureter which needs to be absolutely isolated, varies. In easy cases not more than 1·25 centimetres (half an

inch) need isolation, but with advanced growths 5·0 to 7·5 centimetres (two or three inches) may be needed. The surgeon should always keep the length of absolute isolation as low as possible because of the danger of necrosis. The lower parts of the cardinal ligaments as yet undivided are then severed and the vagina is now entirely free except for its attachment to the pelvic diaphragm.

FIGURE XIII. Dividing the peritoneum on the back of the right broad ligament.

Hæmostasis.—It may be noted that no mention is made of any preliminary clamping of vessels when dividing the cardinal ligaments. The reason is that the cardinal ligaments do not as a rule contain vessels of any size, and clamps applied to them as a routine before division get in the way and prevent the operator from dividing the ligaments as far outwards as possible. It is better to pick up bleeding

vessels in the course of the division as appears necessary. Very often no forceps are required at all.

Dividing the Vagina.—The gauze packing is now withdrawn from the vagina, and the lower limit of the growth having been defined, a Bonney-Berkeley clamp is placed across the vagina sufficiently clear of

FIGURE XIV. Dividing the right cardinal ligament.

the growth, but well above the line where it is intended to divide the vagina. The exact point will differ, but in general the object is to take away as much vagina as possible. By the technique described practically all the vagina is removed, but in early and favourable cases the surgeon may decide not to remove as much as this. The division is made with the scalpel, the bladder and ureters being carefully held out of the way. I divide the anterior wall first, then the two lateral walls, and

finally the posterior wall, taking the greatest care beforehand that the rectal wall is clear of it (Figure XVI).

Securing Vessels.—The operation area having been mopped clear, search is made for bleeding points. As a rule there are not many. Those most likely to give trouble lie about 2·5 centimetres (an inch) lateral

FIGURE XV. Completing the separation of the vagina posteriorly.

to the vagina. The tissue in which they run should be picked up and included in a mattress ligature; indeed, owing to the depth at which the operator is working, it is as a rule best to under-run bleeding points with a needle, as tying them direct is difficult. Obvious bleeding vessels having been secured a swab soaked in 1,000 "Flavine" should now be packed into the pelvis. "Flavine" is something of a styptic and the pressure also helps to check oozing.

Removing the Regional Glands.—The areolar tissue outside the external iliac artery on one side is divided parallel to the artery. The tissue covering the artery and vein is now separated from them inwards towards the brim of the pelvis, carrying with it the glands along the iliac vessels. When the inner edge of external iliac vein is reached,

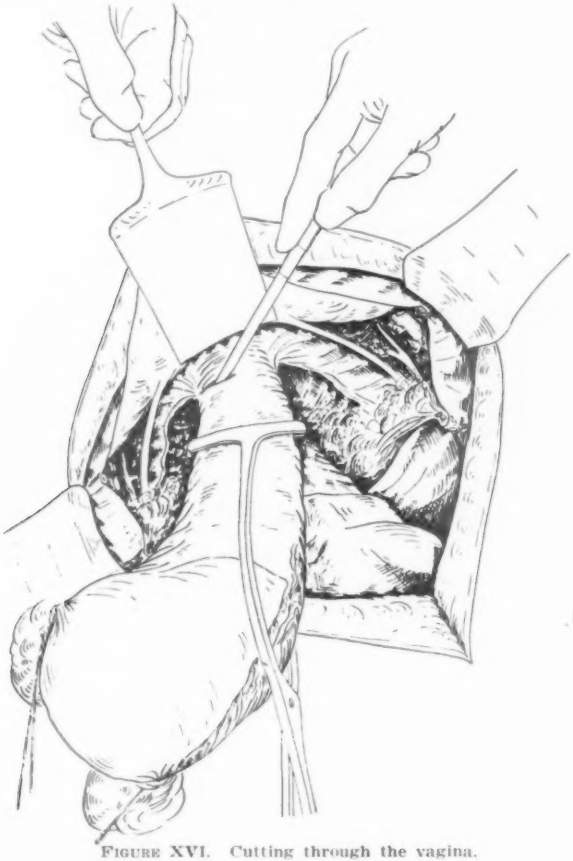

FIGURE XVI. Cutting through the vagina.

this separation is continued down the side wall of the pelvis to the level of the obliterated hypogastric artery. The forefinger is now pushed under the artery and the vessel raised up all the way from its origin from the anterior trunk of the internal iliac artery down to its disappearance at the cornu of the bladder. The artery is then divided at the cornu of the bladder and the whole length of it "rucked back"

to the internal iliac trunk, carrying with it the cellular tissue and glands already separated from the external iliac vessels. A ligature is now placed at its junction with the internal iliac trunk or on that trunk itself, and the mass is cut away, carrying with it the ligature originally placed on the proximal end of the uterine artery (Figure XVII). The extent to which this group of glands should be removed depends on the case. When the external iliac glands are obviously malignant or are believed to be malignant, the common iliac glands must also be removed, and occasionally it is necessary to go as high as the

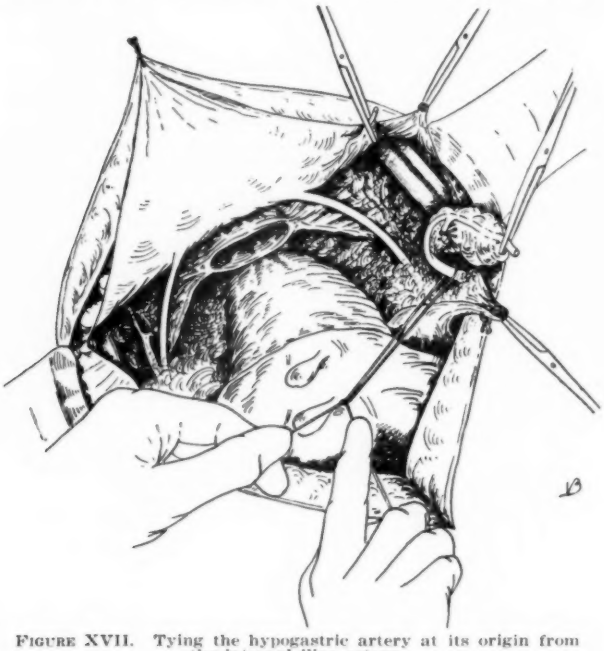

FIGURE XVII. Tying the hypogastric artery at its origin from the internal iliac artery.

aorta. Malignant adhesion of a gland or glands to the external iliac vein is not uncommon, but even then separation can be effected as a rule. Having removed the iliac glands on one side, the surgeon should then turn to the other side and repeat the procedure.

There remain to be removed two very important groups of glands, namely, those occupying the obturator fossæ. The procedure is as follows. The excision of the obliterated hypogastric artery and the sheet of tissue and glands which have been described, lays bare the obturator fossa, in which a mass of yellow fat will be found lying. The forefinger should be inserted into the fossa under the external iliac

vein, and by means of it and forceps this mass should be dislodged. Running through it, is the obturator nerve which should be manœuvred out of it. It will be found that by gentle traction and manipulation the whole of this mass, which contains the obturator glands, can be got cleanly out of the fossa, including a rat-tail-like extension which reaches almost as far as the line of the groin. The fossa should be left entirely empty down to the bare bone of the pelvic side wall (Figure XVIII).

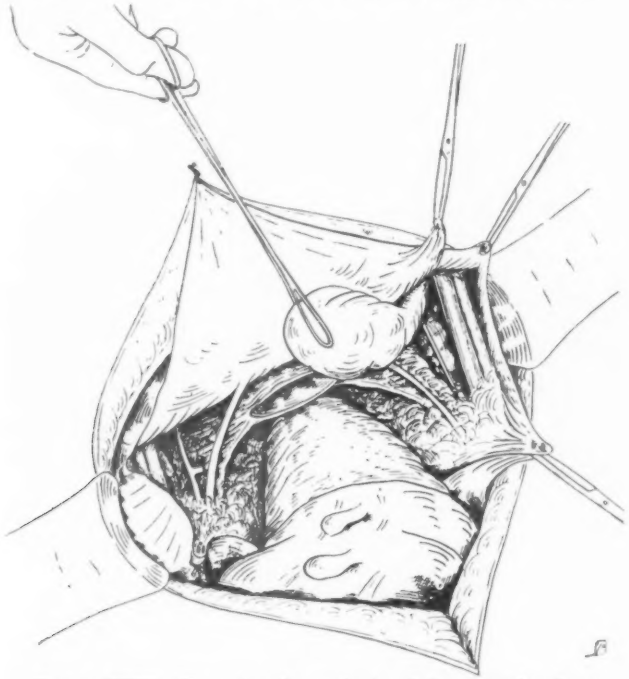

FIGURE XVIII. Removing the contents of the obturator fossa but the nerve left *in situ*.

Packing the Pelvis.—If the vagina has been removed to the extent described, it is usually impossible to suture its open end, nor is it necessary. In the past, when I did not remove so much vagina I used to suture it, but not now. Instead, I pack into the pelvis about two feet of gauze bandage soaked in 1 in a 1,000 "Flavine," the lower end being pushed through to the vulva. The gauze is removed in twenty hours by pulling on that end.

Suturing the Pelvic Peritoneum.—The anterior peritoneal flap, which up to now has remained sutured to the lower end of the abdominal wound, is liberated, and by means of a continuous catgut suture starting at

the point where the left ovario-pelvic ligament was ligatured, is attached to the peritoneum covering the posterior half of the pelvis and the bowel, the suture line ending at a corresponding point on the right side. Care must be taken not to pick up the gauze packing lying immediately beneath the peritoneum or to prick the ureters or iliac vessels, both of which lie uncovered in close relation to the peritoneal edges along which the suture is carried (Figure XIX).

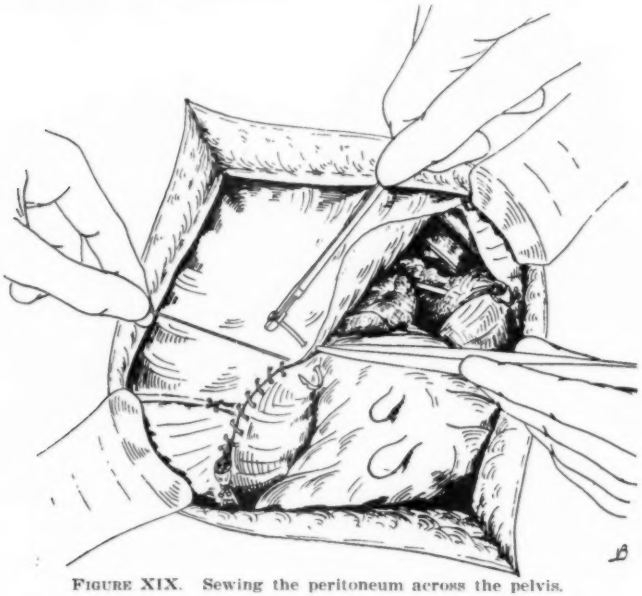

FIGURE XIX. Sewing the peritoneum across the pelvis.

Closure of the Abdominal Wound.—The abdominal wound is now closed. The suturing should be strong, as in patients debilitated by carcinoma the healing powers are below normal.

TREATMENT OF SHOCK.

There is often a good deal of shock, especially in those cases in which troublesome oozing continues throughout the operation, and, moreover, many of the patients are in a poor state to begin with. My practice is to feel the pulse directly the operation is over, and if it is not entirely satisfactory, I administer saline venous infusion before the patient leaves the table. Saline venous infusion is almost as good for shock as blood transfusion, if it is carried out immediately, but the longer it is delayed the less effect it has. Blood transfusion, on the other hand, does not lose in effect nearly so much by being delayed. It is

best, therefore, to do the saline infusion immediately, and reserve blood transfusion for later on in case the patient's condition requires it.

I avoid rectal infusion if I can, because the bowel has been considerably bared and loosened. Continuous saline infusion under the skin is a useful adjunct and camphor in oil hypodermically is the best stimulant to use.

AFTER TREATMENT.

After treatment is that of an ordinary abdominal operation, except in regard to the bladder. If the operation has been carried out in the manner I describe, the bladder is paralysed for a week or more and regular catheterization is a necessity. Even after the patient has begun to pass her urine, the catheter is still required, for the viscus is only partially emptied. Generally it takes from two to three weeks for the patient to obtain full command over her bladder.

REMARKS ON THE OPERATION.

The operation varies very much in difficulty according to the degree to which the growth has advanced. More handicapping than advanced growth, however, is adiposity, which, when extreme, renders the operation exceedingly difficult, for it compels the operator to do a great deal of the work by sense of touch alone.

As regards the duration of the operation, the surgeon should not hurry, for hurried work means rough work and shock. The great thing to be avoided is free loss of blood, and this is best achieved by care and deliberation. There are occasional cases in which continual oozing of blood uncontrollable by ligatures or forceps pressure is present from the start, and in such the surgeon has no choice but to be as quick as possible. Fortunately, these bad cases are rare. My usual time for the operation nowadays is between an hour and an hour and a quarter, though I have performed it in as little as twenty-seven minutes at a stage in my experience when I set more store on speed than on meticulous accuracy of performance.

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(1) Victor Bonney: "The Results of Wertheim's Operation and a Comparison Between Them and Those Obtainable at Present by Radium," *Proceedings of the Royal Society of Medicine*, July, 1929, page 1313.

(2) Victor Bonney: "Hunterian Lecture on the Surgical Treatment of Carcinoma of the Cervix," *The Lancet*, February 8, 1930, page 277.

THE ASSOCIATION OF OVARIAN NEOPLASMS WITH ENDOMETRIAL HYPERPLASIA.

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IN recent years the changes occurring in the endometrium, both in normal and abnormal states, have deservedly received considerable attention.

Any organ which undergoes such extraordinary modification in normal circumstances as does the endometrium, necessarily presents greater problems in the elucidation of the abnormal conditions. This applies particularly to those states which are dependent on the action of the still imperfectly understood hormones. Nevertheless, recent experimental research has gone far to clarify many of our difficulties and clinical and pathological observations lend supporting evidence for some of the hypotheses.

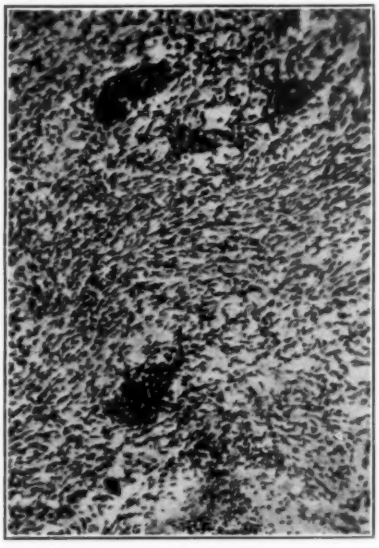

FIGURE 1. Photomicrograph of a section of the tumour described in Case I. Groups of cells resembling those of the *stratum granulosum* merge into stroma-like cells. × 150.

The actions of secretions arising from various structures in the ovary have been investigated experimentally, and some have been determined. Robinson[41] has sought to associate some ovarian tumours with and to derive them from definite epithelial structures, considering that the effect of the secretions of both of them on the endometrium is, in part, evidence of the similarity of their nature.

Numerous cases have been reported in which certain varieties of tumours and cysts of the ovary have been associated with either endometrial proliferation or the growth of endometrial glands in abnormal situations.

Before considering these neoplasms, it is necessary to examine those normal constituents of the ovary which have an influence on the endometrium. That the ovary has a powerful internal secretion (originally thought to be one entity) was shown by the work of such investigators as Limon,[25] Tuffier,[53] Morris[35] and Steinach.[48] Recent research indicates that the ovarian hormone comprises several constituents.

It has been recognized for some time that the *corpus luteum* has a very potent effect on the endometrium. The experiments of Loeb showed

FIGURE II. Photomicrograph of a section similar to that shown in Figure I. × 150.

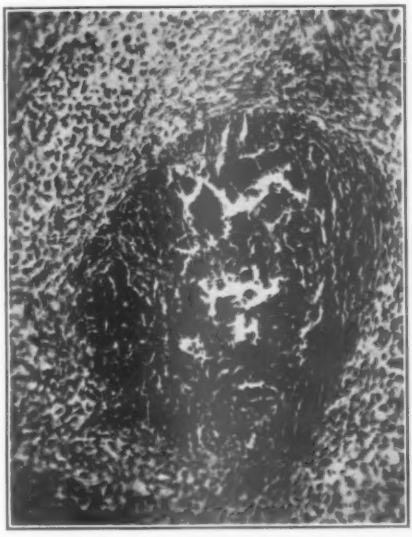

FIGURE III. Photomicrograph of another portion of the tumour shown in Figure I. *Stratum granulosum* cells merge into stroma-like cells. × 150.

that it was responsible for the embedding of the ovum, and he demonstrated that the luteal body was necessary for the decidual changes, since these either did not occur or retrogression took place if the ovary was removed or the *corpus luteum* cauterized.[27] The premenstrual changes also have been shown to be due to a substance produced by the "false" *corpus luteum* of menstruation.

More recently, experiments performed on animals show that the Graafian follicle produces a secretion which is similar to that of the *corpus luteum*, and has a powerful effect on the endometrium and the vaginal epithelium.[1] [2] This material may be found in follicular cysts and has been obtained at operation even in human subjects.[3]

The experimental work has thus demonstrated that a secretion of the Graafian follicle, folliculin, almost entirely replaces in its action that of the whole ovary.

It has been shown that various stages of development of the endometrium may be correlated with the various stages of development of the follicles. Thus the cells of the Graafian follicle and the *corpus luteum* are responsible for the changes in the endometrium during the menstrual cycle.

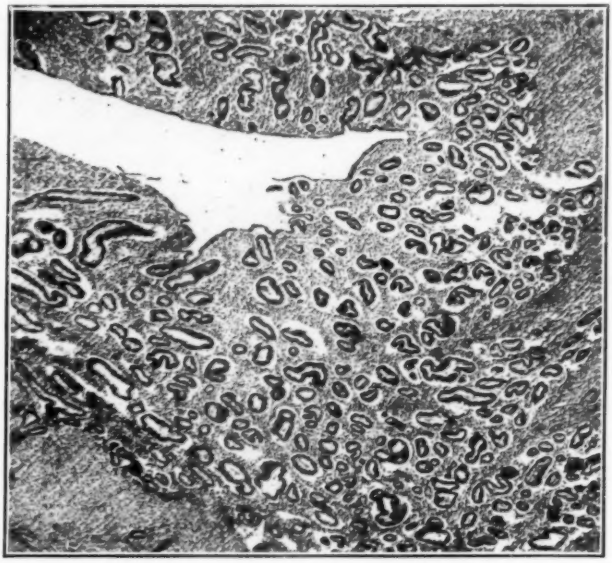

FIGURE VI. Photomicrograph of the lining of the uterus (Case II) showing the marked hyperplasia present. × 40.

We may now consider some pathological aspects of the problem of the relationship of pathological ovarian conditions and alteration in the endometrium.

It has been shown that a persistent luteal cyst may be associated with and presumably produce an hypertrophy of the endometrium.[12] The amount of luteal tissue in the walls of some of the cysts may be quite large, so that probably it is the excess secretion which is responsible for the abnormality present.

There are other cases in which the relationship is not so obvious. Schroder[47] and Tietze[52] have described tumours of the ovary arising from the granulosa cells of the Graafian follicle, with an accompanying glandular hyperplasia or aberrant growth of endometrium.

In these cases it is possible that the cells in question were producing hormone, which is similar to that ordinarily produced by the normal Graafian follicle. It is obvious, however, that some other factor must be at work in these cases, determining the exact site and distribution of the proliferation; but, at the same time, it may be inferred that the endometrial development is due, in part at least, to an influence derived from the tumours of the *stratum granulosum*.

Other cases have been described in which the tumours were considered to be mesenchymal in origin, and unless we postulate an irritant effect on the surrounding organ (compare on the analogy of glandular hyperactivity, the thyreoid, for example, in the presence of foreign tumours), it is difficult to imagine the connexion between the ovarian tumour and endometriosis. This is discussed subsequently and an alternative explanation suggested.

The writer[21] has described recently three cases in which hyperplastic and heterotopic endometrium was associated with tumours of the ovary. In two cases the tumours were of the granulosa cell type, while in the other the tumour arose in a luteal cyst.

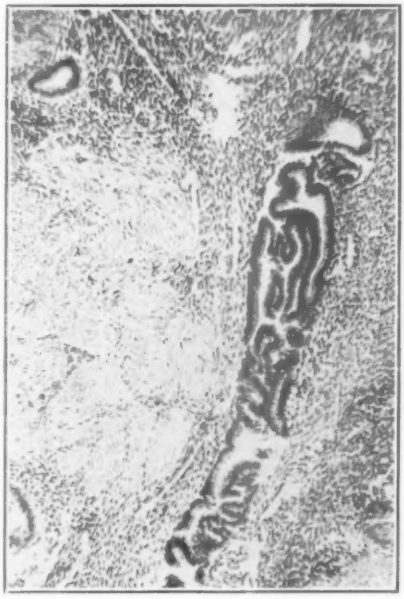

FIGURE VII. Photomicrograph of a portion of the wall showing an "endometrial gland" present in the wall (Case II). × 110.

CASE REPORTS.

The following further cases are reported because the ovarian tumours with which the endometrial proliferation was associated, were composed almost entirely of stroma tissue.

CASE I.—Mrs. R.S., *ætatis* 59, complained of hæmorrhage *per vaginam* for three years prior to her seeking treatment. Her previous history showed nothing noteworthy. The menopause occurred at the age of forty-eight.

On examination she was found to have a mass in the lower portion of the abdomen in the mid-line and extending towards the left side. On vaginal examination the uterus, which was displaced forward and to the right side by the growth, appeared to be normal. The tumour occupied the left fornix and the pouch of Douglas. At operation a large solid tumour developing from the region of the left ovary was removed, together with the uterus. For six months after operation the patient was well.

On pathological examination the tumour was oval in shape, measuring 10·0 by 11·25 by 15·0 centimetres (four by four and a half by six inches); it had a smooth surface and was solid. On section it showed a variegated appearance, white, yellowish, reddish and brownish areas being intermingled. Some portions showed the characteristic marbling of the "sarcomata" of the ovary. A few small cystic spaces were present.

On microscopical examination rounded areas of epithelial cells (resembling the cells of the *stratum granulosum*) were present, merging at the periphery into typical stroma cells (see Figures I and II). In some cases the cells between the two varieties closely resembled *theca interna* cells. Some of the "epithelial" cells formed cords and tubules. A few small cysts resembling atretic follicles were found.

The uterus was normal except for some hyperplasia and hypertrophy of the endometrium. This was markedly in excess of the epithelium normally present in the organ of a woman of her age.

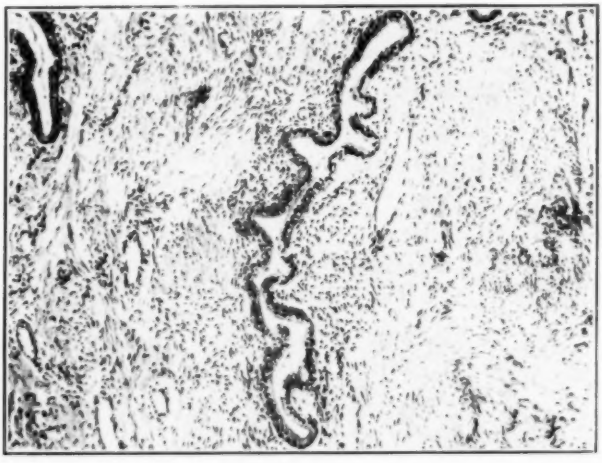

FIGURE VIII. Photomicrograph of a portion of the adenomyoma of the recto-vaginal septum showing a "gland" space amongst the muscle tissue (Case II). × 110.

CASE II.—Mrs. R.A., *ætatis* forty-seven, had been married twenty-six years and had one child, twenty-five years of age. There had been no other pregnancy. She complained of severe menorrhagia for the previous twelve months, though, prior to this, there seemed to have been some excess at the periods for a considerable (undetermined) time. Recently there had been occasional pain in the groins during the menstrual periods. There had also been a "weighing down" feeling and dysuria. A tumour had been observed in the hypogastrium for about six months.

On examination of the abdomen a tumour was felt in the mid-line of the hypogastrium and a second to the right of this. On vaginal examination the uterus was enlarged and a mass was felt projecting from the back of the cervix into the recto-vaginal septum. There was also the large mass felt in the right fornix bimanually.

At operation hysterectomy was performed and a tumour occupying the position of the right ovary was removed.

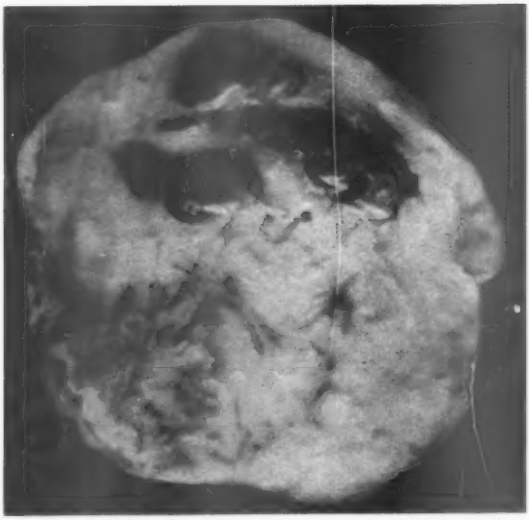

FIGURE IV. Colour photograph of the tumour of the ovary (Case II). The tumour measured 15 by 15 by 10 centimetres (six by six by four inches) and was a typical stromatogenous neoplasm.

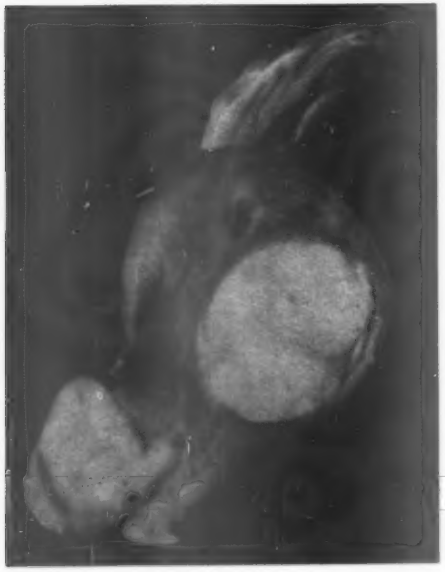

FIGURE V. Colour photograph of the uterus (Case II) showing a myoma of the body and an adenomyoma of the recto-vaginal septum.

Pathological examination showed that the uterus was irregularly enlarged and measured 12·5 by 10·0 by 8·75 centimetres (five by four by three and a half inches). On section (see Figure V) it showed a large myoma and a post-cervical myomatous nodule.

The tumour replacing the right ovary measured 15 by 15 by 10 centimetres (six by six by four inches). On section (see Figure IV) it showed a more or less homogeneous appearance, at one part cystic. On microscopical examination the tumour of the ovary presented very cellular tissue, with some degenerated areas. This cellular tissue was very similar to normal ovarian stroma, the cells being very closely packed, having spindle or irregular nuclei and almost no protoplasm and very little supporting tissue (Figure IX). In some areas small cystic spaces which closely resembled atretic follicles were present.

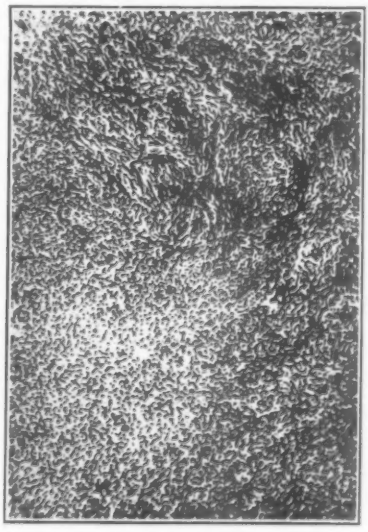

FIGURE IX. Photomicrograph of a portion of the tumour (Case II), illustrating the resemblance to ovarian stroma. × 45.

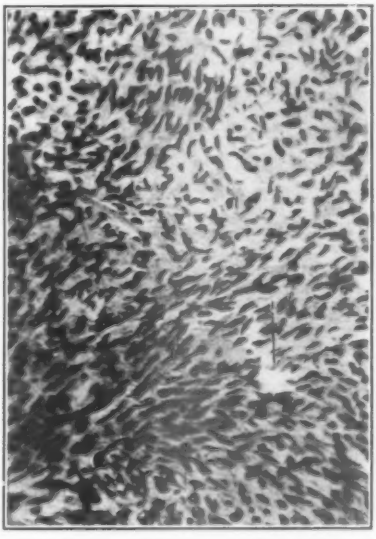

FIGURE X. Photomicrograph of another portion of the tumour. × 180.

The endometrium of the uterus was very hyperplastic, though there was no evidence of inflammation (see Figure VI). The glandular tissue extended into the wall of the uterus for a short distance (see Figure VII). The myoma was a typical leiomyoma and contained no glands. The "myoma" in the post-cervical region contained glands of an endometrial type continuous with those of the mucous membrane as in Cullen's cases.[10] [11]

CASE III.—Mrs. E.E., *ætatis* sixty-two, complained that she had had slight vaginal hæmorrhage at intervals for ten years. This had been severe for seven days prior to admission. Her previous history was uneventful. The menopause had been at the age of fifty-one.

On examination the uterus was somewhat enlarged; a polypus projected from the *os uteri*; there was a mass in the region of the right ovary. The patient was admitted to hospital. The polypus was removed and diagnostic curettage was performed.

Microscopical examination of the polypus and scraping revealed typical adenocarcinoma (see Figure XIV).

Laparotomy was performed and the uterus, which was the site of several myomata, was removed, together with a tumour of the right ovary. The patient made an uneventful recovery. Deep X ray therapy was subsequently given.

Pathological examination showed that the uterus measured 8·75 by 7·5 by 5·0 centimetres (three and a half by three by two inches), and contained a number of small myomata. One myoma, measuring 3·75 centimetres (one and a half inches) in diameter, projected from the posterior surface and was pedunculated. On section, an oval mass of tissue 1·8 by 2·5 centimetres (three-quarters by one inch) projected into the cavity, arising from the posterior wall. Microscopically the tissue projecting into the cavity of the organ was an adenocarcinoma. There was some invasion of the muscle by the growth in its neighbourhood, but the greater part of the wall and the myomata were free from glands.

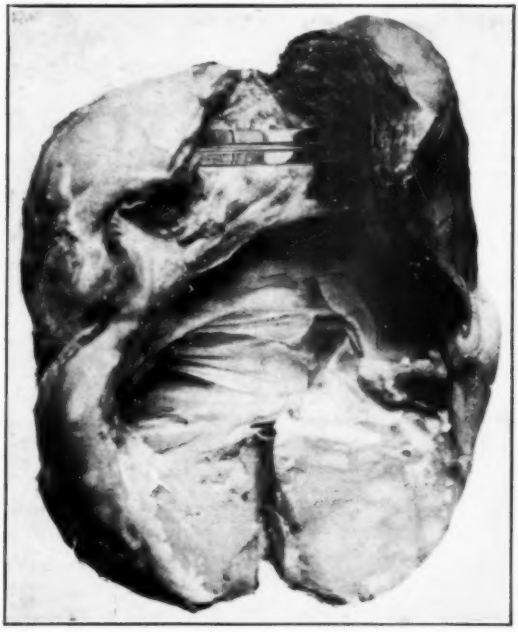

FIGURE XI. Photograph of the tumour of the ovary (Case III). There is a large degenerative cyst present. The tumour measured 10·0 by 6·75 by 5·0 centimetres (four by two and a half by two inches).

The ovarian tumour had an irregular smooth surface, measured 7·5 by 6·25 by 5·0 centimetres (three by two and a half by two inches) and was mainly solid. It contained an irregular cavity, apparently degenerative (see Figure XI). Microscopical section showed the tissue to be similar to that of ovarian stroma. No follicular-like structures were observed.

DISCUSSION.

In these examples the ovarian tumours were associated with definite abnormalities of the endometrium. In Case I there was merely considerable hyperplasia and in Case II not only local hyperplasia, but invasion of the muscle coat and the formation of an adenomyoma of the recto-vaginal septum occurred. The relationship of the endometrial neoplasm in Case III is problematical, but since the metrorrhagia had been present for years, in all probability there had been for some time endometrial hyperplasia in which malignancy had arisen recently. The return of the endometrium to normal after removal of the tumour in some cases where the uterus has not been removed, lends support to the contention that

there is a definite relationship between the ovarian and endometrial conditions.

The association of endometrial variations with diseases of the ovary, particularly neoplasms, has been described by numerous writers.[6] [13] [22] [23] [34] The majority of the cases in which the ovary is involved have presented an explanation, for example, luteal cysts have been present. In other examples the tumours have been genetically related to the epithelial structures of the organ, for example, in the case of the granulosa cell tumours.[18] [37] [45] [46] [52]

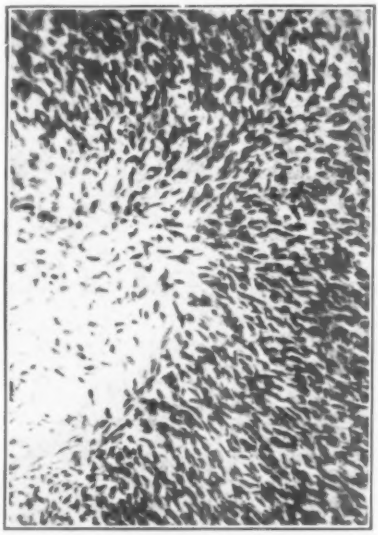

FIGURE XII. Photomicrograph of a portion of the tumour (Case III). The tissue resembles the stroma of the ovary. × 180.

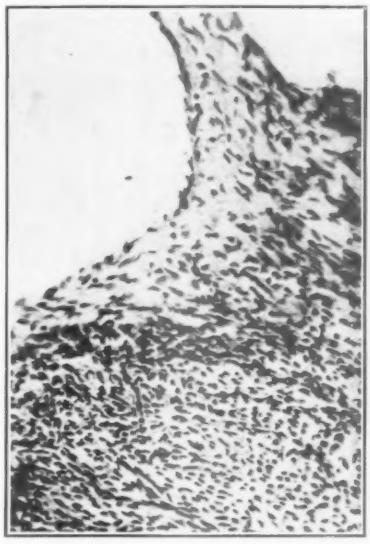

FIGURE XIII. Photomicrograph of another portion of the tumour (Case III). × 110.

In the examples described here, however, the tumours are derived apparently from the stroma tissue of the ovary. Although in the case of the epithelial tumours the relationship presents but little theoretical difficulty, correlation of endometrial hyperplasia with the presence of these stromatogenous growths calls for some special explanation. Three possibilities suggest themselves:

1. The presence of the growth may cause irritation of and perhaps excess secretion from the epithelial components of the ovarian tissue which remains.

2. Epithelial elements may be present in some portions of the tumours which have not been examined.

3. The stroma of the ovary may be closely related to the epithelium and by structural and functional activity possibly have a similar effect on the endometrium.

1. The suggestion that the presumable hormone arises in the portion of remaining normal ovary is without any evidence. Undoubtedly an insignificant portion of ovarian tissue suffices for the maintenance of normal function, as demonstrated by those cases in which both ovaries appear to be replaced completely by, for example, "dermoid" cysts. Nevertheless direct evidence in favour of this contention of irritation of ovarian tissue by a tumour is not forthcoming.

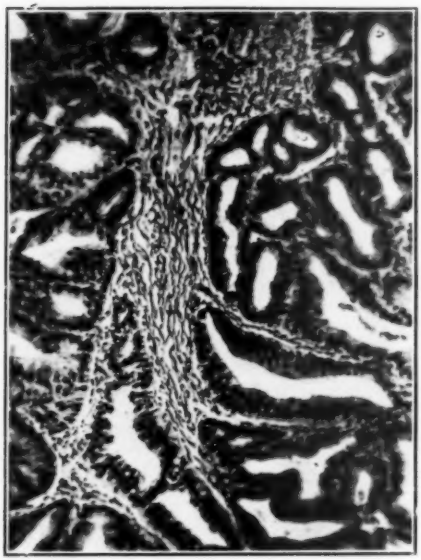

FIGURE XIV. Photomicrograph of a portion of the scrapings taken from the uterus in Case III. × 90.

2. In so far as complete histological investigation of a tumour is seldom performed owing to its size, the presence of epithelial elements in some parts of the growth conceivably may be overlooked. In most cases, however, all characteristic areas or portions differing in macroscopical appearance are examined. This problem, however, merges into the next one, the relationship of the stroma cells to the epithelial structures of the ovary.

3. The ovarian tumour described in Case I affords a clue not only to the relationship of these growths to the endometrial abnormality, but also to the nature of the stromatogenous tumours themselves. It consisted of groups of cells resembling those of the *stratum granulosum* and around these and merging into them were the stroma cells. Some "*theca interna*" cells were also present.

In discussing the carcinomata of the ovary, Ewing states:

> In a well-defined group the cells are usually small, and grow in indistinct alveoli or groups which vary in size. Such tumours may present areas of diffuse growth, and . . . . the cells become intimately mingled with the stroma. . . .

In some typical examples of granulosa cell tumours I have observed the gradation of the epithelial cells into stroma cells. The discovery of granulosa cells or of structures resembling atretic follicles in some of the stromatogenous tumours is not an uncommon finding.

Observations such as these suggest strongly that the stroma is closely related to the epithelial structures of the ovary. The stroma is

described usually as being mesenchymal in origin, whereas the "epithelium" is, of course, mesothelial in its characters.

From a consideration of the embryology, histology and physiology of the ovary, I have been led to the opinion that the stroma cells of the organ are distinct from connective tissue of mesenchymal origin and distinct from the connective tissue of the ovary itself. The stroma arises from the germinal nucleus, that is, the mesothelium, and is closely related to the "epithelial" structures. The evidence for this view will be discussed in detail in another paper. It would appear, however, that the stromatogenous tumours of the ovary may be responsible for local and heterotopic endometrial hyperplasia in a manner similar to the "epithelial" growths. Whether this interpretation or the hypothesis of stimulation of the portion of remaining ovarian substance be more correct, remains for further observations to determine.

FIGURE XV. Photograph of the uterus (Case III) showing the adenocarcinoma arising from the endometrium. Above the growth the endometrium is thickened. An incision has been made in the tumour and endometrium.

One question of importance, when considering the value of an observation or series of observations such as these quoted, is the large number of cases in which such association of tumours and endometrial change does not occur.

Three possibilities arise: (i) The cells of the growth may not be functioning. (ii) There may be other factors at work in the body preventing the action of a secretion of the tumour. (iii) Observations may not be complete.

The third possibility is the one to which I would call attention. The absence of gross menorrhagia or metrorrhagia does not mean that endometrial change is not present. Many cases of ovarian growths, even with minor menstrual irregularities, are treated without investigation of the endometrium.

On the other hand, unexplained menstrual disturbance should lead to an examination of the ovaries for some change and gross pathological change occasionally will be encountered. The suggestion that endometrial

hyperplasia (as shown by microscopical examination of curettage material) might be due to an ovarian tumour, has resulted, in a few cases in my experience, in investigation for and discovery of such a growth.

Thus, though the association of the tumours of the ovary of stromatogenous nature and the endometrial proliferation is established for the cases described, careful investigation of all cases of this and related types is necessary to further our knowledge.

SUMMARY.

1. Three tumours of the ovary of the stromatogenous variety, each of which was associated with abnormality of the endometrium of the uterus, are described.

2. One of the tumours appears to be a mixed granulosa cell and stromatogenous tumour, thus linking up these last with the granulosa cell tumours proper.

3. Various endometrial changes—local hyperplasia and heterotopic proliferation—were found.

4. Hypotheses concerning the association are discussed.

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METASTATIC TUMOURS IN THE INTESTINES.

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The alimentary tract, despite a large aggregate blood supply, is rarely the seat of blood-borne metastatic growths. Serosal involvement of the intestines is, of course, frequent in peritoneal carcinomatosis of ovarian, gastric or other origin; but true hæmatogenous secondary tumours in the gut wall itself are unusual. The four examples to be described in this paper occurred in a consecutive series of 250 autopsies on malignant neoplasms of all kinds, an incidence of only 1·6%. Malignant melanoma certainly exhibits a greater capacity than other kinds of tumours for producing secondary growths in the intestine. Thus Eiselt[7] found bowel metastases in twelve of fifty cases of this disease, an incidence of 24%; and for a large proportion of recorded secondary neoplasms of the gut melanotic tumours are responsible. Of this character are cases described by Askanasy,[1] Bland-Sutton,[4] Girdwood,[8] Hada,[9] Kaufmann,[12] Robb,[17] Ross,[18] Virchow,[22] and others. In a study of metastatic growths in the spleen, di Biasi[3] records nine cases which exhibited also bowel metastases, and of these no less than six were melanotic or pigment-free nævus carcinomata, while two were from breast cancer, and one was from carcinoma of the submaxillary salivary gland. Metastases to the bowel from primary tumours of the pharynx, uterus, stomach and thyreoid are described by Kaufmann,[12] who elsewhere[13] also records jejunal deposits from carcinoma of the prostate. Simpson,[20] Barnard and Elliott[2] and Maxwell and Nicholson[14] record intestinal deposits from bronchial carcinoma; Borrmann[5] from cancer of the œsophagus and from cancer of the breast; Israel[10] from gastric carcinoma; Kantorowicz[11] from cancer of the breast; Schirokogoroff[19] from a small round-celled tumour of the pancreas; and Prym[16] from chorion carcinoma. B. Müller[15] describes a case with hæmangiomatous growths in gums, pleura, lungs, bones, thyreoid gland and bowel; while inclining to the view that the condition was a "system disease," he admits the possibility of metastasis from one of the tumours as a primary growth. Teacher[21] saw bowel metastases which he regarded as intestinal implantations from a gall bladder tumour *viâ* a patent cystic duct, an interpretation open to serious doubt. Davis[6] mentions an instance of intestinal metastases from carcinoma of the male breast, but gives no particulars.

The present paper is to record four examples of blood-borne metastases to the intestine, in three cases from primary carcinomata of the breast, and in one case from carcinoma of the lung.

CASE RECORDS.

CASE I.—The patient, A.D., was a female, aged seventy-four years.

History.—In February, 1928, a small mass was noticed in the left breast. There soon appeared firm enlargement of the left axillary glands, and a little later of the right axillary glands also. Repeated X radiation at a public hospital appeared temporarily to arrest the growths, which, however, in November began to enlarge rapidly. In April, 1929, intermittent attacks of abdominal pain and

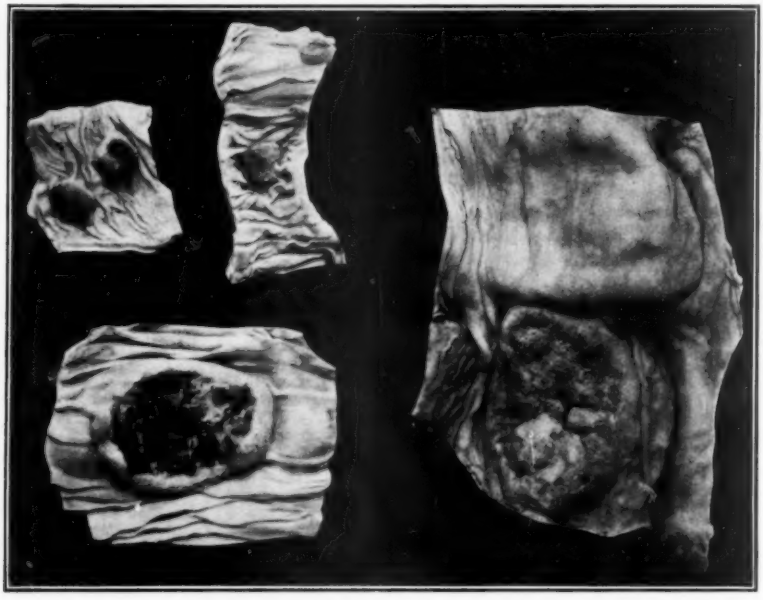

FIGURE I. Intestinal metastases in Case I, viewed from mucosal aspect. Several small pedunculated growths and two large ulcerated ones are depicted.

vomiting developed and hepatic enlargement was noted. A month later a few isolated skin nodules had appeared as far caudally as the right loin and buttock. In June, 1929, signs of right basal pleurisy appeared and death occurred a few days later.

Autopsy.—The body was moderately well nourished. Many button-like non-ulcerated skin nodules up to two centimetres in diameter were scattered over the left breast and scapular regions, and a few similar nodules were present in the abdominal wall, right loin and buttock. Axillary glands of both sides and right supraclavicular glands were prominently involved.

The thoracic cavity exhibited malignant infiltration of the right basal parietal pleura, with recent fibrinous pleurisy over the middle and lower lobes of the right lung. The left lung and pleura were normal. No growths were found in either lung. The heart was not involved. There were deposits of growth in the anterior mediastinal lymph glands, but none in the posterior mediastinum.

The liver, spleen, kidneys, pancreas, stomach and pelvic viscera contained no growths. The perirenal fat at the caudal pole of the right kidney contained an isolated metastasis three centimetres in diameter. Both adrenal glands contained discrete secondary growths. The small intestine contained many metastatic tumours presenting on the mucosal aspect (Figure I). Some of these were small pedunculated nodules projecting into the lumen of the bowel. Others formed oval indurated ulcers with raised margins, the largest measuring four by three centimetres in diameter. Approximately half-way down the small intestine was an intussusception twelve centimetres long, with a pedunculated tumour three centimetres long and two centimetres wide at the apex of the intussusceptum. This was the largest of the pedunculated metastases. The mesenteric lymph glands contained many large deposits of growth.

The skull, brain, meninges and thyreoid gland were uninvolved. None of the long bones was sectioned, but there was no external evidence of metastases.

Histology.—In all situations the tumour was an anaplastic *carcinoma simplex*, with the cells arranged partly in large clumps and partly diffusely. Many giant tumour cells were present, and mitotic figures were plentiful. The smaller bowel nodules occupied the submucous and mucous layers only, the subjacent muscularis being intact and no evidence of the growth being visible from the serous aspect. In the larger ulcerated growths, a variable degree of infiltration of the muscle was present. The submucosa just peripheral to the growths exhibited a zone of linear infiltration several millimetres wide. No subserous infiltration was seen.

CASE II.—The patient, M.C., was a female, aged fifty-eight years.

History.—In May, 1925, the patient first noticed a mass in the left breast. This slowly enlarged, but was neglected until April, 1929, when there appeared right facial paralysis, paræsthesiæ of the right side of the face, and a little later diplopia and right-sided deafness. Investigation at this time disclosed the following abnormalities: a large firm mass in the left breast; signs of paralysis of the ophthalmic division of the fifth and paralysis of the sixth, seventh, eighth, ninth, tenth and twelfth right cranial nerves; nystagmus in all directions, and a staggering gait with falling to the right side; cerebro-spinal fluid under increased pressure, but otherwise normal; no reaction to the Wassermann test; and skiagraphically visible areas of bony destruction in parts of the skull. While in hospital the patient also began to suffer from persistent headaches and vomiting, and she died in July, 1929.

Autopsy.—A firm irregular mass, ten centimetres in diameter, occupied the outer half of the left breast and was adherent to the skin and infiltrated the subjacent muscle. There were no macroscopic deposits in either the axillary or supraclavicular lymph glands. Both lungs contained small scattered metastases, one of which had invaded the lumen of a main tributary of the right pulmonary vein. The bronchial and posterior mediastinal glands were involved. The auricular myocardium contained five small metastases, and one nodule was present in the wall of the right ventricle. Several ribs contained tumour deposits.

The liver, spleen, kidneys, stomach and pelvic viscera were uninvolved. Both adrenals were largely replaced by metastatic tumours. The pancreas contained several secondary growths. The mucous aspect of the small intestine presented eight tumour deposits, some of which were sessile or pedunculated nodules, while others were indurated oval ulcers with hard raised edges, the largest of which was 1·5 centimetres in diameter. Opposite several of these growths the serous aspect of the gut wall presented flat subperitoneal buttons of growth, seen on section to be continuous with the tumours within the bowel. The mesenteric

and cœliac lymph glands contained many large growths. Several discrete nodules were present in the omentum. The large intestine was intact.

The brain contained numerous metastases, both in the cerebrum and cerebellum. The largest of these involved most of the right half of the cerebellum, the *brachium pontis*, the tentorium, and adjacent part of the petrous bone. Extensive neoplastic destruction of the left posterior part of the calvaria was present. The cervical organs were normal.

Histology.—In most situations the tumour exhibited the structure of a solid alveolar *carcinoma simplex*, but in parts of the pulmonary metastases it consisted of disorderly adenocarcinoma with conspicuous areas of mucoid change. Mitoses were not numerous. The bowel tumours, histologically of the *carcinoma simplex* type, involved the muscular as well as the mucous and submucous coats, but extended most widely in the submucosa. In those tumours which involved also the serous coat, there was a zone of peripheral subserous infiltration similar to but less extensive than that in the submucous tissues.

CASE III.—The patient, S.N., was a female, aged fifty-eight years.

A full report of this patient has already been published,[31] and only a brief *résumé* is necessary. The neoplasm was a highly anaplastic carcinoma of the breast with metastases in the lungs, myocardium, liver (one small nodule only), kidneys, pancreas, intestines, *dura mater*, brain, skull and thyreoid gland. One of the metastases in the myocardium had penetrated into the cavity of the left ventricle, the apex of which contained a friable mass of tumour thrombus.

FIGURE II (Case III). Ulcerated metastases in upper part of jejunum.

The condition of the intestines was remarkable. The first coil of jejunum contained three malignant ulcers with hard raised edges, the largest three centimetres in diameter (Figure II). Many smaller ulcerated growths and pedunculated tumours were present in the next sixty centimetres (two feet) of bowel. Ninety centimetres (three feet) from the commencement of the small intestine there was an intussusception ten centimetres long, at the apex of the intussusceptum of which was a pedunculated tumour four centimetres in maximum diameter (Figure III). The next 150 centimetres (five feet) of bowel presented several small growths, and then occurred a second intussusception eight centimetres in length, due to a pedunculated growth three by four centimetres in diameter (Figure IV). A few small nodules were present in the remaining coils of ileum. The ileo-caecal orifice projected prominently into the caecum, suggesting the commencement of an ileo-colic intussusception, and the caudal lip of the orifice was the seat of an irregular pedunculated, slightly ulcerated tumour nearly two centimetres in diameter (Figure V). One small pedunculated metastasis projected into the lumen of the ascending colon (Figure VI). The remainder of the colon was intact. The root of the mesentery contained two lymph glandular deposits of growth each two centimetres in diameter. The sigmoid mesocolon contained a small metastasis. The abdominal lymph glands and peritoneum elsewhere were uninvolved. With the foregoing bowel findings may be related the intermittent attacks of abdominal pain and distension with vomiting which occurred during the last two weeks of the patient's life.

Sections of the bowel tumours showed that the smaller nodules lay entirely in the submucous and mucous layers, the muscularis being uninvolved. In most of the larger pedunculated tumours there was slight infiltration of the muscle, which was drawn up as a conical peak apparently by the traction of the pedunculated mass (see Figure VII, E). The potentialities of such a condition in the production of an intussusception are obvious. Only in the largest of the ulcerated growths was there any extension of the tumour cells to the subserous tissues, and then only over an area much smaller than that of the mucosal ulcer.

CASE IV.—The patient, F.B., was a male, aged forty-four years.

History.—This patient had had a right radical mastoid operation in 1914, and a discharging ear ever since. At the end of December, 1930, he began to suffer from backache, loss of weight, occasional vomiting, and a "thumping" sensation in the head, and his mentality became a little dulled. Examination early

FIGURE III (Case III). Longitudinal section of an intussusception, showing the pedunculated growth X at the apex of the intussusceptum Y.

in February, 1931, disclosed bilateral papillœdema more marked on the right side and the cerebro-spinal fluid, otherwise normal, was under increased pressure. Cerebral abscess was suspected and operation was performed on February 11. A polypus-filled mastoid antrum was found, but no evidence of any bony or intracranial suppuration. Subsequent to the operation there were persistent headache and increasing stupor, and signs of complete left-sided hemiplegia, including the face, appeared. Death occurred on February 28.

Autopsy.—A large nodular mass of firm white growth occupied the hilum of the right lung, invaded the pericardium, the lumen of the superior *vena cava*, and also one of the main tributaries of the pulmonary vein, and was confluent with massive lymph glandular deposits in the posterior mediastinum. The superior mediastinal and lower deep cervical glands also were involved. The left lung and the heart were normal.

Both adrenal glands were largely replaced by metastatic tumours. The third part of the duodenum contained a flat nodule of growth, four millimetres in

diameter, situated in the mucous and submucous layers and not involving the muscle. The upper sixty centimetres (two feet) of jejunum contained two more tumours, the larger one centimetre in diameter and pedunculated into the gut lumen. Both of these were in the antimesenteric part of the bowel wall, and there was no external evidence whatever of their presence. The remainder of the intestinal tract was normal. The root of the mesentery contained several lymph glandular deposits, and the cœliac and upper preaortic glands also were involved. The stomach, pancreas, liver, spleen, kidneys, bladder, prostate and testes were uninvolved.

FIGURE IV (Case III). The second intussusception. The intussuscipiens V has been opened longitudinally, exposing the growth X. Y is the entrant intussusceptum.

The thyreoid was normal. The brain contained a solitary large metastasis, three centimetres in diameter, situated at the junction of the right occipital and parietal lobes of the cerebrum and involving both cortex and white matter. An extensive area of brain around the tumour was unusually soft and diffluent. No other brain deposits were found and the meninges were normal. There was no external evidence of metastases in any portion of the skeleton.

Histology.—All of the tumours were large angular-celled *carcinoma simplex*, the arrangement of the cells strongly resembling prickle-cell carcinoma, but no definite prickles or cornification were found. Mitotic figures were abundant. The intestinal nodules were restricted largely to the submucosa, the subjacent muscularis being entirely free and the mucosa presenting only slight central destruction.

DISCUSSION.

The hæmatogenous route of origin of the intestinal metastases described is attested by: (i) the situation of the younger growths in the mucous and submucous layers in all four cases, a distribution incompatible with lymphatic extension from the serosa; (ii) the absence of adjacent serosal infiltration in all save the more advanced growths; (iii) an obvious source of malignant systemic emboli in three cases, invasion of pulmonary veins in Cases II and IV and intracardiac invasion in Case III; (iv) the presence of widespread remote metastases in other regions. In Case I there were no visible pulmonary metastases,

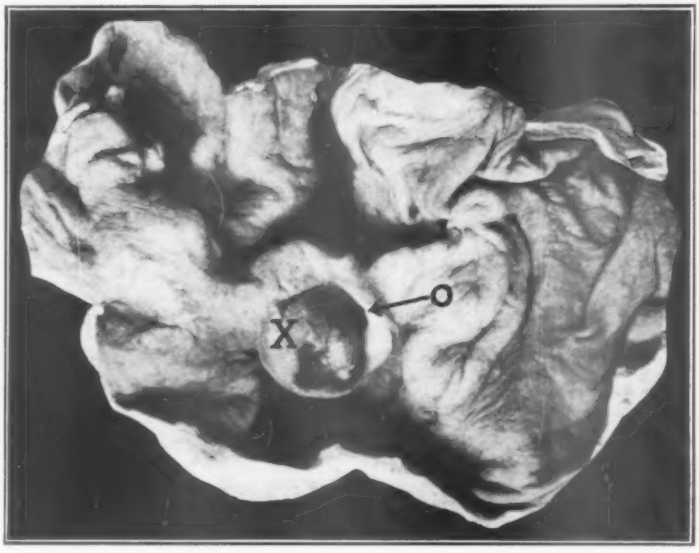

FIGURE V (Case III). The caecum has been opened along its lateral border, exposing the ileo-caecal orifice O, on the lip of which is a pedunculated metastasis X.

and lymphatic extension from the primary growth had produced skin nodules over a wide area; nevertheless the multiplicity and mucosal situation of the bowel tumours and the presence of discrete metastases within the adrenals indicate the blood-borne character of the secondary growths. In all four cases the mesenteric gland deposits were probably secondary to those in the bowel itself.

The relationships of the metastases to the four coats of the gut wall are indicated in Figure VII, which epitomizes the various conditions described in the foregoing reports. The youngest nodules appear always to be situated initially in the submucous tissues, forming a flat sessile button of growth with a peripheral zone of submucosal infiltration (A).

From this stage the tumour may invade both the mucous and muscular coats and produce an ulcerated lesion (B). Further extension through the muscularis may result in the formation of a subserous nodule opposite the ulcer (C). On the other hand, there may occur no gross ulceration of the early nodule, which then produces an enlarging pedunculated mass projecting into the bowel lumen (D). Adhesion of such a tumour to the subjacent muscle may then result in peaking of this coat by traction (E), a condition which constitutes an incipient intussusception.

A very striking pathological feature of all four cases was the escape of the liver from metastatic involvement. As is well known, the liver is the favourite site of prolific secondary tumours following the systemic liberation of malignant cells. Yet in my four cases, despite abundant dissemination and despite the presence also of multiple growths in the portal drained viscera, the liver was entirely uninvolved in three instances; and in Case III, in which widespread systemic embolism was of very gross degree, the only hepatic metastasis was a solitary nodule only two millimetres in diameter. Many of the records already cited from the literature are of interest in this connexion. In all of di Biasi's[3] nine cases which exhibited bowel metastases, widespread deposits in many other tissues were present; yet in no less than five of the nine cases the liver was not involved. This is the more remarkable since in di Biasi's cases there were metastases in both the bowel and spleen; so that in addition to the systemic dissemination of tumour cells there were also multiple portal sources presumably capable of discharging malignant emboli to the liver. In the case described by Israel,[10] despite an extensive primary carcinoma of the stomach and multiple intestinal deposits, there were no metastases in the liver. Concerning his case of carcinoma of the pharynx, Kaufmann[12] makes special comment on the absence of secondary growths in the liver, although metastases were numerous in most other organs. Kaufmann's case of prostatic cancer also,[13] while presenting widespread visceral deposits, did not affect the liver. In both cases of lung cancer described by Barnard and Elliott[2] the liver was uninvolved, although their second case exhibited many secondary tumours in other organs. Girdwood's case of generalized melanomatosis[8] presented secondary tumours in almost all parts of the body, but there was none in the liver. Schirokogoroff's pancreatic tumour[19] had produced metastases in the lungs, kidneys, spinal cord and ganglia, stomach and intestine, but the liver was uninvolved. Müller's angiomatous tumours[15]

FIGURE VI (Case III). Pedunculated secondary growth in the colon.

were situated in many organs, but not in the liver. In twenty-seven autopsy cases reviewed in this paper and of which details are given concerning the systemic distribution of visceral metastases, these were present in the kidneys in fourteen cases, pancreas thirteen, heart twelve, brain eleven, spleen ten, thyreoid ten, adrenals ten, liver ten (in one case of which, however, my number III, only one tiny nodule was present), stomach mucosa six, and other regions less frequently. These figures are in striking contrast to the usual preeminent vulnerability of the liver to disseminated growths in general. Notable is the high incidence of metastases in organs which are decidedly infrequent sites of secondary growths, such as the pancreas, heart and thyreoid. It would appear that systemically sown tumours capable of establishing metastases in the intestines exhibit on the one hand more than average capacity for successfully colonizing certain other "unusual" metastatic sites, and on the other hand are relatively ineffective in colonizing hepatic tissue. These observations support the view that the development of metastatic growths in various tissues depends at least in part on the suitability or otherwise of those tissues as soils for the proliferation of arrested malignant emboli.

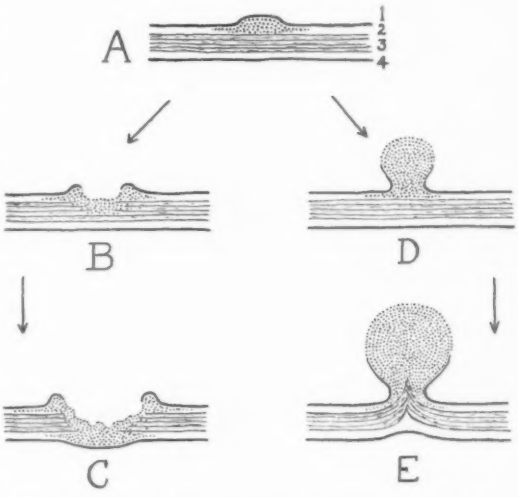

FIGURE VII. A diagrammatic representation of the relationship of metastatic deposits to the four coats of the intestine. Growth tissue is represented by stippling. 1, mucosa; 2, submucosa; 3, muscularis; 4, serosa. A, young metastasis situated in submucosa. B, an ulcerated growth, involving the muscularis. C, a larger ulcerated tumour, extending through the muscularis and infiltrating the subserous tissues. D, a pedunculated metastasis involving only mucous and submucous layers. E, a pedunculated growth adherent to and producing traction invagination of the muscularis and dimpling of the serosa.

The subject under discussion is of practical clinical importance for the following reasons:

1. Secondary growths in the bowel may occasion serious diagnostic errors by producing intestinal obstruction, while the primary growth remains symptomatically latent. Such were Bárnard and Elliott's first case and one of those mentioned by Bland-Sutton.

2. After a short or long period following the apparently successful eradication of a primary melanotic growth, a solitary metastasis in

the bowel may necessitate operative interference. Of this nature were Robb's two cases and Ross's case.

3. In patients suffering from malignant tumours, unsuspected intestinal deposits may cause prominent clinically puzzling obstructive symptoms. This was exemplified in my Cases I and III. Obstruction is due most frequently to the formation of an intussusception by a pedunculated growth.

SUMMARY.

Four examples of blood-borne metastatic tumours in the intestines are described, and a number of other records from the literature are reviewed.

Features of pathological and clinical interest regarding such growths are discussed.

ACKNOWLEDGEMENTS.

I am indebted to members of the Honorary Staffs of the Austin and of the Alfred Hospitals, Melbourne, for access to the clinical records of patients under their care.

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THE PEPTIC ULCER PROBLEM.

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THE problem of gastric and duodenal ulceration has been of continued interest and dissension since the first case was recorded in 1586 by Marcellus Donatus, of Mantua. With gradually increasing intensity the records of medicine and surgery have been filled with reports, until today the literature reaches enormous dimensions. It is my endeavour to put before you in this address some of the advances made in the knowledge of this subject, and to select from them information which may throw light on the causation and treatment of peptic ulceration.

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS.

It would be well in the first place to recall the anatomical conditions which have direct bearing on this subject and the disturbed physiological conditions which influence pathogenesis and healing.

It is unnecessary to describe the anatomy of the normal stomach or the structure of its walls. It is, however, important to point out that the weakest portion of the muscle coats of the stomach is in the fundus and cardiac region. This is probably the reason why the rare true diverticulum appears in this region (see Figure I).

If bursting pressure is applied to the whole stomach the first part to rupture is the fundus, next the cardiac region and the last area to give way is the pyloric region and the duodenum. Of all the coats of the stomach the mucosa and the *muscularis mucosæ* have the greatest resistance to distension. This property is of prime importance in insuring the safety of the Rammstedt operation and of plastic operations on the pylorus not involving the mucosa.

The different types of stomach and variations from the average will not interest us in this paper and will not be further discussed. It is well to recall the fact, however, that gastric ulceration occurs in the stomachs of average length, very rarely in short stomachs and frequently in long stomachs. Duodenal ulcerations rarely occur except in persons with short stomachs with the greater curvature running diagonally across towards the pylorus high above the umbilicus. In Hurst's view, whenever a duodenal ulcer is found with a large stomach it is *prima facie* evidence of a pyloric block being present (Figure II). The enlargement is really secondary to such a block, and with appropriate treat-

ment the stomach returns to its normal condition. Moynihan claims that the stomach never properly regains its normal size. It is certainly true that the mucous membrane does not become normal, for it is always thrown into much deeper folds when the muscle wall retracts (Figure III).

The blood supply of the mucous membrane of the lesser curvature has been shown by Reeves to be less than that of the rest of the stomach.

FIGURE I. True diverticulum of posterior wall of stomach near lesser curvature. Oblique view. (Dr. A. J. Reye.)

The vessels are more tortuous, smaller, and do not anastomose so freely there as elsewhere. This arrangement of vessels also appears in the first 2·5 centimetres (one inch) of the duodenum, and is noticed particularly on the anterior surface, which has been called Mayo's white spot. This area is rendered more distinct by the application of slight traction on the duodenum, and is the site of the majority of duodenal ulcers.

The muscle tonus of the stomach varies with the volume of the contents to maintain a pressure of between four and six millimetres of mercury, with the result that the upper level of the gastric contents is the same whether they measure one or two pints.

The lesser curvature has a comparatively small amount of movement, and as a result the mucous membrane in that area is seldom, if ever,

FIGURE II. Penetrating duodenal ulcer with pyloric block causing dilatation of the stomach. (Dr. V. McDowall.)

flattened in the same manner as that of the rest of the stomach. The folding of the mucosa in this situation is longitudinal and the channel has been named by Aschoff the "gastric pathway." It is along this pathway that food first travels before being churned in the stomach.

The peristalsis is nearly continuous, as the stomach is empty for only a few hours in the early morning. As each peristaltic wave

approaches the pylorus, the pylorus relaxes somewhat, but the canal remains very narrow, and the acid chyme in fluid state is squirted, under pressure, into the duodenum. Relaxation of the pyloric sphincter is inhibited whenever the mucous membrane of the vestibule comes in contact with anything such as insufficiently masticated or very hot or

FIGURE III. Another and later picture of Figure II, showing deep folds of mucous membrane which has not retracted in the same ratio as the musculature. The usual enlargement of the jejunum distal to the anastomosis is well shown. (Dr. V. McDowall.)

very cold food, which might injure the duodenal mucous membrane by its mechanical properties.

The gastric juice, containing 0·4% hydrochloric acid and pepsin, is secreted by the glands of the pylorus and body of the stomach. The accompanying diagram shows the distribution of the glands in the stomach (Figure IV). The pyloric portion forms three-tenths of the whole of the stomach, and there is a transitional area of about one centi-

metre between the pyloric region and that containing the oxyntic cells. The oxyntic cells are scantily found throughout the whole of the greater curvature.

The glands of the pyloric vestibule secrete an alkaline fluid containing no digestive ferment and probably identical with the duodenal secretion. Special cells in all parts of the stomach secrete mucus, the glands of the fundus being regarded by McCann as being of special importance in secreting mucus to combine with a constant fraction of the changing volume of acid secreted. The regurgitation of duodenal contents is not of any great importance in the reduction of the acidity of the gastric juices. McCann performed experiments on dogs to prove this statement. He shunted the duodenal contents altogether away from the pylorus to a distal portion of the ileum. The removal of the duodenal juices in no way affected the acidity of the gastric juice.

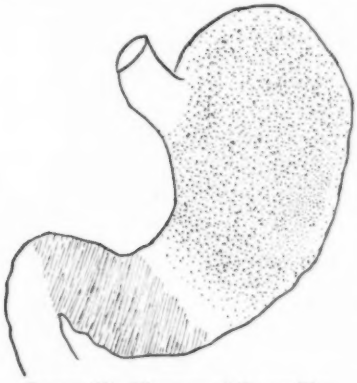

FIGURE IV. Diagram of the position of the oxyntic cells. They extend roughly for two-thirds of the lesser and three-quarters of the greater curvature. The acid secreting area is stippled, the alkali secreting area shaded.

I do not propose to describe the normal secretion of gastric juice, but would remind you that the products of digestion and meat extractives, absorbed from the intestine after the latent period of one or two hours, cause a flow of juice in the last stages of gastric digestion. There is also resting juice secreted independently of these factors, which varies in quantity from two to fifty cubic centimetres per hour. It is this resting juice, in disturbed physiological conditions, which is the cause of a great deal of damage to the already injured mucosa. Any treatment for ulceration must be planned with full cognizance of its presence and must include measures to neutralize it.

X ray examination has shown that the normal duodenal bulb is more or less filled throughout a meal, whereas the rest of the duodenum is never full, the food passing rapidly through it. The bulb would seem to be more of an annex to the stomach, with somewhat similar functions and, like it, subject to peptic ulceration. The rest of the duodenum is really the proximal segment of the small intestine. A small quantity of chyme, squeezed through the pylorus at each peristaltic wave, collects in the duodenal bulb. No churning effect takes place here and no mechanical rubbing of its membrane such as occurs in the pyloric antrum. Hurst suggests that the absence of this mechanical irritation may be the reason for the rarity of cancer in this position as compared with its frequent occurrence in the pyloric vestibule. The secretion of the duodenal membrane is alkaline and is quite insufficient to neutralize the acid chyme in the normal stomach. This gastric chyme seldom remains in the first

part of the duodenum for much more than half a minute. It is rapidly neutralized in its passage through the rest of the duodenum.

It is generally recognized that the resistance to peptic digestion is gradually diminished from the stomach to the intestine, and this fact must always be remembered in the operative treatment of ulceration and particularly in conditions associated with a high gastric acidity.

The gradation of resistance accounts for the greater frequency of duodenal ulcers over gastric ulcers and the tendency to form jejunal ulcers. It is generally agreed that the further down from the duodenum an anastomosis is made to the stomach, the greater the risk of occurrence of jejunal ulcer. For instance, jejunal ulcer follows anterior gastro-jejunostomy twice as often as it does the short, no loop, posterior gastro-jejunostomy.

From experiments performed by McCann[1] of the Mayo Clinic, it would appear that the gastric mucosa possesses considerable immunity to the autolytic activity of the normal acid and pepsin, and that the duodenal mucosa when robbed of its alkaline protective juices is rendered very liable to peptic ulceration.

ÆTIOLOGY.

Mechanical injury by coarse cellulose food is a prominent factor in the initiation of ulcer, and it seems that we must now pay still greater attention to diet in post-operative treatment.

It is now generally accepted that the conditions necessary for the production of a peptic ulcer depend on the presence of acid and pepsin, the initial lesion being always dependent upon a preceding gastritis or duodenitis. Peptic ulcers found in other situations are dependent upon the presence of gastric mucous membrane (gastric heteropia). The situations in which ulcers of this kind have been reported are: (i) The pharynx, generally bilateral on the posterior wall. (ii) The lower end of the œsophagus. Chevalier Jackson reports fifty-one cases in this situation. (iii) The second part of the duodenum. I have seen one at the junction of the second and third parts of the duodenum, penetrating the pancreas. (iv) Diverticula of the small intestine: these ulcers are a big factor in the tendency of diverticula to perforation. Such inflammatory conditions are regarded as being the results of blood infection, but damage to the mucous membrane by coarse cellulose foods and secondary surface infections must now be admitted as a primary cause.

The incidence of associated dental affections with peptic ulcer of all types is shown by Spencer-Payne[2] to be 74%. The percentages for the different types are as follows:

Gastric ulcer	63%
Duodenal ulcer..	74%
Gastro-jejunal and jejunal ulcer	78%

These infections include *pyorrhœa alveolaris* and apical infections, and a combination of the two. Naso-pharyngeal infections reported by

Gill-Carey include infected tonsils and sinuses and reach the incidence of 29%.

Associated abdominal conditions are undoubtedly a factor to be considered, but the difficulty here is to decide whether they are pathogenetic factors or only part and parcel of a general intestinal infection.

It is such a common experience to find evidence of infection in the glands of almost any part of the mesentery, mesocolon, gastric and gall bladder areas, that it is difficult to regard any one of these abdominal infections as a separate entity. It is of interest to note that with the exception of the glands in the mesocolon these enlarged glands are found almost always on the right side of the abdomen. It is conceivable that infections attacking the intestinal canal and its associated organs would naturally select for attack the area or areas crippled by some form of dysfunction so that any combination of vulnerable points may be found.

I have in my mind a man of sixty-five years on whom I operated with the intention of performing a gastro-jejunostomy for a duodenal ulcer. At operation we found a large inflamed ulcer of the duodenum, a gall bladder full of stones, a large, hard, swollen pancreas, a long white appendix and adhesions around the terminal part of the ileum (caused by an old hernia) producing a mild partial obstruction. The only thing I did not do was gastro-jejunostomy. With simple medical treatment and advice in regard to diet by his physician he is perfectly well today, some four years after his operation.

This patient illustrates a condition in which most of the infections associated with ulcer are present.

The incidence of associated abdominal conditions has been studied and reported on by Hartman and Rivers[3] from records of 1,075 cases, with ulcer found at operation. The percentage reported by them is as follows:

Gastric ulcer, no abdominal complications	50·8%
Chronic appendicitis	35·7%
Subacute appendicitis	3·1%
Duodenal ulcer	5·2%
Cholecystitis	2·1%
Carcinomatous change	2·1%

You will observe the high incidence of appendicitis, the weakest organ of the abdomen, and the absence of definite associated disease of other organs in only half the cases.

Moynihan first drew attention to the mimicry of gastric and duodenal ulcer by disease of the appendix, and pointed out that a great number of patients who were adjudged to be suffering from ulcer were really victims of appendicitis.

Later on it was found that removal of such an appendix did not always give permanent relief, that symptoms too often recurred and that local or epigastric disturbances returned almost immediately or within a few months and then the ulcer symptoms again made their appearance. A number of patients with a history broadly falling into that group were reoperated on and in a surprisingly large number no ulcer or lesion to explain their symptoms could be found.

Right-Sided Ptosis.

Wilms[4] in 1908 first pointed out the high incidence of undue mobility of the caecum in unsatisfactory operations for appendicitis for which no other explanation could be found. His paper attracted a good deal of attention and the subject soon acquired literature of its own.

Waugh's paper in 1920[5] marks the stage of real advance in our knowledge of this subject, but owing to the lack of adequate explanation for his theory and clinical experience, most surgeons regarded his claims with intolerant disbelief. His clinical results compare favourably with most of the standardized modern surgical procedures.

Carslaw[6] in 1928 published a paper on this subject and was able to deal with the ætiology and trace the production of symptoms from the pathological condition in a much more satisfactory manner. Since his paper appeared reporting clinical results, the subject has assumed a much greater significance. Herein lies the reason for the failure to relieve the right-sided pain associated with appendicitis in apparently clear-cut cases of that disease, and also the explanation of the symptoms of dyspepsia diagnosed wrongly by skilful physicians as diseases of the stomach, duodenum or gall bladder.

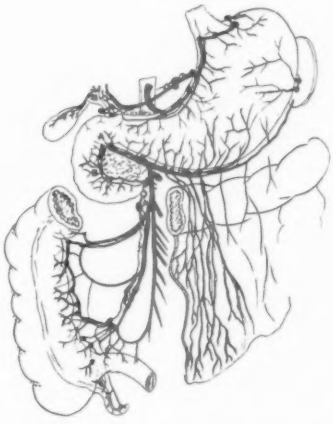

FIGURE V. Lymphatic distribution from ileo-caecal area to cæliac glands. (Braithwaite.)

Waugh and Carslaw both drew attention to the very high incidence of coloptosis with inflammatory conditions of the stomach, duodenum and gall bladder. For two years I have adopted the practice of noting the presence or absence of right-sided ptosis in any case of ulcer or gall bladder disease. The intention has been to get some idea of the value of their statements as applied to my own practical experience, and I can confirm as far as one can in that short period of time, the high incidence of right-sided ptosis in diseases of the gall bladder and in peptic ulcer.

The condition on which I place most stress is one in which downward traction of the ileo-caecal area puts tension on the superior mesenteric artery and its accompanying veins and lymphatics; when the bowel is pulled down the weight is mainly taken by the superior mesenteric artery. The condition gives the feeling that the ileo-caecal portion of the bowel is swinging on the artery, and it sometimes stands up from the mesentery as much as 18 millimetres (three-quarters of an inch). It is nearly always accompanied by a row of glands of different sizes extending up to the lower border of the third part of the duodenum (Figure V).

It is difficult to account for the reflex symptoms by a pull of ordinary intensity exerted on that area of mesentery, and for a satisfactory explanation a much more sensitive path must be found to explain the disturbed motility and secretion. That some such disturbance does occur I have no doubt, but the difficulty arises in estimating just what degree of ptosis and what arrangement of conditions are necessary to produce a mechanical pull sufficient to cause sympathetic disturbances. Ptosis of the worst degree can be present without producing any pull at all on the artery or any sympathetic disturbance, and moderate degrees may be present in which the whole weight of the caecum swings on the artery. In one instance in which the whole ascending colon was in the pelvis, any downward pull was taken by the underleaf of the mesentery and no traction at all could be transferred to the artery. This woman had none of the usual epigastric symptoms. The higher the stomach, duodenum and gall bladder are placed, the more likelihood there is of a mild degree of right-sided ptosis producing symptoms.

A proper appreciation of the condition which causes these symptoms is necessary for its treatment, and its correct valuation is essential in an attempt to associate this condition with ulcer as a causative factor or as a condition interfering with satisfactory treatment. It has also undoubtedly to be considered as a condition likely to cause trouble in diagnosis. I believe that right-sided ptosis has to be seriously suspected as a pathogenetic factor in a big proportion of peptic ulcers, and I am certain that it is the cause of many failures in treatment by modern surgical measures.

To illustrate this particular aspect there is the case of a man of thirty-seven, a dredge-hand, who suffered for many years with severe epigastric pain and who got no relief of his symptoms by medical treatment. His gastric analysis resembled that of a duodenal ulcer, and his X ray examination revealed a persistent filling defect at Haudeck's angle on the lesser curvature.

A large, thickened gastric ulcer was found at operation and a post-gastro-jejunostomy was performed with dramatic relief of pain. He has had no return of pain in any form since, but has had recurrent attacks of severe vomiting lasting from one to three days and occurring about every three weeks. He feels perfectly well in between attacks and has maintained his weight.

The caecum and ascending colon were noted at operation as being down in the pelvis and his peculiar attacks of vomiting were ascribed to that condition.

His abdomen was reopened and a colopexy performed. The anastomosis was in clean working order, and his ulcer had disappeared, leaving only a little white scarring on the peritoneum. He is perfectly well now as the result of the colopexy and is rapidly gaining in weight.

This man illustrates a condition in which the ulcer was cured, but the symptoms of dyspepsia in peculiar form remained. These were the results of the right-sided ptosis, and had no relation to the gastric ulcer or to the anastomosis.

To attach ptosis with significance to the ulcer problem it is necessary that we consider it from three different angles: Mechanical, nervous (sympathetic), and infective.

Mechanical Causes of Ptosis.

Ptosis is a developmental failure of proper rotation and the descent of the colon. If you care to notice such conditions, you will be rewarded by finding a remarkable variation in the type and in the degree of fixation, the details of which are to be found in Waugh's article. When only isolated bands of fusion are present, they may partly take the pull of the weight of the colon, and it is in these conditions that the mechanics of the condition are difficult to estimate. Beyond calling your attention to such conditions, I will not deal further with them.

Sympathetic Disturbance and Ptosis.

Professor H. Woollard[7] has written an authoritative summary of the state of our knowledge, and perusal of that article will be amply repaid.

The anatomy and physiology of the sympathetic innervation of the stomach, duodenum and intestine have been given quite a lot of attention in the last few years, but our knowledge of the subject is still incomplete and unsatisfactory from the surgeon's point of view.

Alvarez[8] removed strips of stomach and bowel from an executed criminal and as a result of his studies thereon, he considered that the longitudinal and circular muscles and the mucosa of all of the alimentary canal possess the inherent property of rhythmic contraction and relaxation and maintain their rhythm even after five days in the refrigerator. There is good evidence to show that there is a gradation in the latent period from short intervals at the cardia and long intervals at the *pars pylorica* and also some evidence to show a similar gradation from the duodenum to the lower part of the ileum. All the movements of the stomach and bowel persist after the division of all of the extrinsic nerves, and it would appear that their function is limited to regulation and coordination of the alimentary movements as a whole. The muscle of the duodenal cap is sluggish and that of the terminal ileum very active.

Working on dogs carefully trained, Hartzell[9] found that section of the vagus in the thorax produces marked reduction in both free and total acidity. The highest post-operative curve was lower than the lowest pre-operative curve. When the nerves were cut in the abdomen there was no reduction of acidity, but the secreting curve was much shorter and the stomach emptied one to two hours sooner than before section of the vagus.

It has been generally held that the vagus and the sympathetics act antagonistically, the vagus acting as the pressor and the sympathetic as the depressor influence. Alvarez and others, as the result of experiments on rabbits, conclude that at least in the rabbit both vagus and the sphincters served as inhibitors and regulators of intestinal activity. The only sign of antagonism between the sympathetics and the parasympathetics was found in the studies of conduction which was improved by vagotomy and injured by splanchnicotomy.

Splanchnicotomy slowed the latent period in all parts of the bowel, except the duodenum; and the intestinal movement was rendered more active and was so sensitive that, when touched, it contracted so as to form a firm white cord. This sensitive condition of the bowel is of interest in the condition of enterospasm, a case of which I have seen after gastro-enterostomy.

Woollard,[7] quoting Hecht, states that the presence or absence of the mucous membrane affected the movements of the stomach. Thus the presence of the mucous membrane increased the movements to be obtained from a preparation of the body of the stomach, while its presence inhibited movements of the preparation of the pylorus. Removal of the mucous membrane reversed these effects.

Thus it might be concluded that Meissner's plexus is predominant in the movement of the body of the stomach and Auerbach's plexus in the pylorus. The relation between these movements is much affected by intragastric pressure.

The pyloric control is by a reflex starting from the mucous membrane and conveyed to the muscle by the intrinsic nerve plexuses. The acid control theory is not now feasible.

Gray held the view that the vagus acts as a power behind active peristalsis, powerful in the stomach and gradually losing its influence as it approaches the terminal part of the ileum. He regarded the sympathetic as the inhibiting factor, influential in the colon and without effect by the time it reached midway along the stomach.

We could then assume that section of the sympathetic supply to the colon would lead to more active movements and more rapid evacuation, and Wade,[10] of this city, has demonstrated that such effects do follow ramisection by Royle's method in Hirschsprung's disease.

So far the results of operative treatment of the nerves of the stomach give very little information of value. Mayo has cut the vagus in 25 cases and reports symptomatic relief and 25% reduction of acidity.

Nazaroff[11] injected the nerves of the stomach in the lesser curvature with alcohol and wiped the main arteries of the stomach with alcohol and reports the removal of pain in all cases, including four of cancer of the stomach.

Schiassi[12] divided the nerves of the stomach in six cases of gastric ulcer with symptomatic relief in all six.

D'Arcy McCrae[13] reported twenty cases; he sectioned one vagus without effect, but double vagotomy caused retardation of function, reduced tone and delayed the emptying time. Splanchnic section gave him the opposite effect.

Fraser[14] groups the effect of sympathetic stimulation under four headings: (i) Those cases in which there appears to be an abnormal sympathetic activity leading to contraction and inducing the state of spasm. (ii) A continuation of the state of spasm inducing achalasia. (iii) Persistence of inhibitory effects leading to atony. (iv) Anomalous nervous activity leading to irregular peristalsis and disturbed motility.

Altogether the problem of the control of the movements of the intestinal canal is in a state of confusion, and with such evidence no theory can be fully established. There is one centre from which we might expect further information and that is from the University of Sydney, the home of so much original work on the sympathetic nervous system.

Action of Sympathetic Nerves on Blood Vessels.—Otfried Müller[15] in 1922 was impressed by the observation that patients with gastric ulcer were afflicted with "vasoneurotic diathesis," a congenital and often inherited condition of dysharmony in the structure and activity of the peripheral blood vessels. The arteries, capillaries and vessels of the skin and especially of the mucous membrane of the lips, showed the greatest irregularity when examined by the capillary microscope. In some places they were contracted, and in other places dilated into varices. This spastic-atonic condition must give rise to great irregularity in the blood supply and a tendency in some areas to stasis. In 1924 he applied his observations to the gastric mucous membrane, and studied the warm, fresh specimens from thirty-two gastrectomies for ulcer of the stomach and duodenum. He found the same condition in the vessels of the stomach as he found in the mucous membrane of the lip in every one of the thirty-two examined. These changes were most marked in the lesser curvature and the pyloric vestibule, and especially in the neighbourhood of the ulcer. The condition found in the stomach of patients with duodenal ulcer was equally constant, but not so severe as in those with gastric ulcer. The vascular changes were independent of the age of the ulcer, degree of acidity and presence of gastritis. A similar appearance was found in only five out of eighteen cases of gastric cancer, and in four of these it was thought the cancer developed from an ulcer.

Infection and Ptosis.

The normal flow of lymph from the terminal fifteen centimetres (six inches) of ileum, the appendix and the caecum is by vessels which unite to form a group of five or six efferents passing to the chain of glands dotted along the line of the ileo-colic artery. The main trunks then pass up in front of the third segment of the duodenum into the glands grouped round the origin of the superior mesenteric artery, and from there into the *receptaculum chyli*. The lymph flow from the ileo-caecal angle would normally not reach either the duodenum or stomach. In the living human subject it was found possible to get the dye to pass from the appendix wall, through the ileo-colic chain, to the superior mesenteric group, and also to the subpyloric group,

If there is a chronic lymphadenitis or lymphangitis, the lymph flow may be diverted from the superior mesenteric glands to the right, over the head of the pancreas to the anterior and posterior pancreatico-duodenal glands (which lie along the anastomotic arch formed by the superior and inferior pancreatico-duodenal arteries in the concavity of

the duodenum). From these glands the flow is to the subpyloric group, and thence through the hepatic glands to the cœliac group. Now, if there is obstruction to the normal flow, it is easy to understand how lymph may from the pancreatico-duodenal glands reach the mucous membrane of the duodenum, from the subpyloric glands reach the mucous membrane of the lower part of the pylorus, and from the hepatic glands reach the upper part of the pylorus. Again, the normal flow from the mucous membrane of the lesser curvature of the stomach is through the superior gastric glands (which lie along the coronary artery) to the cœliac group. As these also receive efferents from the hepatic glands, it is easy to understand how by retrograde flow infective lymph may reach the mucous membrane of the lesser curvature from the hepatic glands. Again, the normal lymph flow from the great omentum is to the inferior gastric glands (which lie along the right half of the greater curvature of the stomach), and thence to the subpyloric group. This was demonstrated by Braithwaite,[16] who, laying gauze soaked in the dye on the lower free border of the omentum, found that the dye rose along the blood vessels of the omentum and filled the inferior gastric glands. If, however, there is lymphatic obstruction, lymph may readily be diverted, and, by retrograde flow, reach the mucous membrane of the greater curvature of the stomach.

The effect produced by the arrival of infective material in the mucous membrane of the duodenum and stomach depends, among other factors, on the virulence of the infective agent. Thus, in the case of the duodenum, should the infection be acute, there may be an acute duodenitis with a lymphatic spread to the gall bladder and ducts. If, however, the infection is less acute, there occurs erosion of the lymph follicles of the duodenal wall. This one would expect to find most commonly in the place where there is the greatest collection of lymphoid tissue. This point corresponds to the "anæmic spot" of Mayo, on the anterior wall about 2·5 centimetres (one inch) beyond the pylorus, towards the upper or antimesenteric border. Again, as this area is more exposed to the action of the acid gastric secretion, erosions are less likely to heal than in the distal parts of the duodenum, where the contents are alkaline. The well-established fact that the chronic duodenal ulcers are almost invariably found in the first segment seems to support this theory. Similar conditions are found in the stomach, and, as in the case of the duodenum, one would expect those areas of the mucous membrane which are richest in lymphoid tissue to be most commonly affected. Bolton[17] states that the lymph follicles in the lesser curvature are twice as numerous as and larger than those in the rest of the stomach, and it is in these areas that gastric ulcers most commonly occur.

SURGICAL TREATMENT.

The surgical treatment of peptic ulcer has for its object not only the relief of symptoms, but protection against complications and recur-

rences. It is an art as well as a science, and the most successful methods are dependent upon the proper evaluation of the advantages of the many surgical procedures and a thorough knowledge of their indications.

In 1892 Doyen performed gastro-jejunostomy for the first time on a patient with chronic gastric ulcer and later reported fourteen further successful cases. Duodenal ulcers as a whole are best treated by gastro-jejunostomy, more drastic measures being reserved for isolated cases with special indications.

Gastric ulcer is in a somewhat different catagory on account of its association with cancer of the stomach, and because it is a much more dangerous disease. It is the fear of these complications that has led so many surgeons to adopt the more radical operation of partial gastrectomy.

To assess the advantages of one type of operation over another, large figures and close, accurate follow-up systems are necessary.

Surgery owes a great debt to Moynihan in England and to the Mayos in America, and these master surgeons may with justice be considered the leaders in this branch of surgery.

Duodenal Ulcer.

Moynihan's views on the operation of gastrectomy for duodenal ulcers are interesting and amusing.[18] He says that gastrectomy for duodenal ulcers is totally unnecessary in the very large majority of cases. He is definitely of the opinion that more patients would die of the operation than would acquire a recurrent or a jejunal ulcer after a gastro-jejunostomy. He writes:

TABLE I.

Showing Results in Gastro-jejunostomy in Duodenal Ulcers.

CLASSIFICATION.	PERCENTAGE.
Very Good	67·2
Good	22·3
Fair	5·1
Poor	5·4
Secondary ulceration	2·8

It is one more evidence of the familiar Teutonic capacity for finding a difficult way of doing easy things. I regret that the wise and cautious judgement, the sanity of outlook, and care for the individual patient, which hitherto have always characterized British surgery, have not rendered all our surgeons immune from rash and most intemperate persuasions by Continental and trans-Atlantic operators.

Nevertheless, it must be admitted that occasionally in recurrent ulcers more drastic measures are necessary, such as gastrectomy by the many methods available or by gastric exclusion of the type per-

formed by H. B. Devine, of Melbourne (Figure VI). Before any type of operation is performed it is presumed always that careful search has been made for infective foci and careful investigation by gastric analysis and appropriate medical treatment carried out; and I again want to emphasize the necessity for careful attention to the right colon.

FIGURE VI. Partial exclusion for penetrating ulcer of duodenum (H. B. Devine). Pólya anastomosis. Screen showed first part of meal to run along lesser curvature and quickly drop to lower portion of stomach and rapidly leave by the dilated jejunum. A small portion of the meal ran round the proximal jejunum into the duodenum and then into the narrow excluded portion of the stomach. It rapidly returned to the stomach and does not show in the picture. (Dr. B. L. W. Clarke.)

The operation of posterior gastro-enterostomy properly performed is a highly satisfactory procedure. I will mention only a few points in connexion with it.

The opening should be adequate; that is, when completed, it must be at least twice the width of the diameter of the small intestine. The intestine cannot deliver more duodenal juice to the stomach than the bore of the jejunum and the activity on its muscle walls allow, and the distal portion cannot drain away more stomach contents than its bore and activity permit. Adequate drainage by an opening of that size is dependent on the size of the jejunum. Anastomoses of considerable length, 11·25 to 12·5 centimetres (four and a half to five inches), have been recommended. They give no greater drainage than the size mentioned and, if the openings

are too far apart, there would appear to be a greater risk of a subsequent jejunal ulcer.

McCann's study in experiments of peptic ulcer shows that when the secretions of the duodenum were shunted from the distal side of the

FIGURE VII. Gastro-enterostomy. Note small jejunum. The patient was perfectly well for three years after operation, then severe hæmorrhage occurred. Arrows indicate reverse flow of gastric contents due to faulty technique. (Dr. B. L. W. Clarke.)

pylorus into the fundus of the stomach, ulcers formed on the anastomosed jejunum in 80% of the experiments, in spite of the fact that the whole of the duodenal contents were mixed with the gastric juice.

Wilkie[19] recommends that the site of the anastomosis should be

below the œsophageal opening, so that full drainage may be obtained. Sherren and Patterson[20] recommend that the anastomosis should be made above the level of the incisura in cases of gastric ulcer. My experience of this procedure, when applicable, has been very satisfactory. Other men claim that unless anastomosis is made at the most dependent portion of the stomach, drainage, the essential part of the operation, is unsatisfactory.

It is always to be remembered that the higher the acidity, the greater the risk of jejunal ulcer and the greater the necessity for post-operative alkaline and dietary treatment.

The unsatisfactory cases are those in which the jejunum is of small bore (Figure VII). The patients with large jejunums appear to have little immediate trouble after operation, most probably because the stomach is effectively drained.

Gastric Ulcer.

The treatment of a gastric ulcer has divided surgeons into two camps: those who recommend partial gastrectomy for all cases of gastric ulcer in which the operation is feasible, and those who rely on an anastomosis (see Tables II, III, and IV).

Balfour[21] reports that 50% of gastric ulcers in the Mayo Clinic are unsuitable for a partial gastrectomy. These are presumably the worst cases from the point of view of size and situation, but he claims that the results justify the procedure. Moynihan and Pauchet[22] and Bastianelli[23] report highly successful results with partial gastrectomy, and favour that operation.

The size of an ulcer is important when we are considering the results of treatment, and from the reports it would appear that many of the ulcers urgently requiring removal are left *in situ*. In the Leeds series of 156 cases, 85% measured less than 2·5 centimetres in diameter, and in the Mayo Clinic series of 638 simple gastric ulcers, 93·8% measured less than 2·5 centimetres.

We must now consider the incidence of cancer because of its bearing on the method of treatment. Wilson and McCarty[24] in 1919 claimed that 71% of cancer of the stomach showed sufficient evidence of previous ulcer to warrant placing them in a group labelled carcinoma developing in ulcers. Conversely, 68% of resected ulcers were associated with cancer. Since that time there has been a gradual fall in reported estimates.

Spilsbury concludes that probably no more than 1% to 2% of peptic ulcers become cancerous. On clinical grounds very diverse opinions have been expressed. Osler found only 2·6%; German statistics of 1,700 cases showed 3·4%; Stewart placed the percentage at 6·1.

When figures are searched in the after-history of patients who have been subjected to gastro-enterostomy for simple ulcers, it is found that the development of malignant disease is uncommon. Patterson in 1914 reported 1% with malignant change; Rufus in 1924 reported 1% to 2%. Balfour shows that among people operated on for gastric ulcer

the death rate, in the first few years after leaving hospital, was three times as high as among the general population.

In a series of 1,280 patients with gastric ulcers operated on during the fifteen years period 1906-1920 inclusive, 195 patients were dead

TABLE II.

From the British Medical Association Investigation, Showing Results of Treatment of Pyloric Ulcers (Probably Duodenal Ulcers).

CLASSIFICATION.	PERCENTAGE.
Very Good	76·3
Good	16·2
Fair	4·1
Poor	3·4
Secondary ulceration	0·9

No carcinoma reported.

TABLE III.

From the British Medical Association Investigation, Showing Results of Treatment of Gastric Ulcers.

CLASSIFICATION.	PERCENTAGE.
Posterior gastro-enterostomy with excision caut or enfolding	Very good results 91·7
Posterior gastro-enterostomy only	50·9
Partial gastrectomy	63·6

No authentic cases of carcinoma were reported.

TABLE IV.

Showing Results of Treatment in Gastric Ulcer by Gastro-jejunostomy with or without Resection.

SURGEON.	MORTALITY.	CURE RATE.
Walton	2%	90%
	2·9%	92%
Sherren		90%
Mayo Clinic		85%

after satisfactory recovery from the operation. Of these deaths 40% were due to cancer of the stomach. No fewer than forty out of fifty deaths among those treated by conservative methods occurred within

two years of operation, suggesting that in some at least, probably in most, the condition at the time was one of cancer.

I have never seen cancer develop in the stomach after gastro-enterostomy at the site of simple ulcers, nor have any of my colleagues in Brisbane with whom I have discussed the matter; and I know that there are at least some busy surgeons in Sydney who have had the same experience. In ten years no patient with cancer of the stomach, following gastro-enterostomy, has been admitted to the Mater Misericordiæ Public Hospital in Brisbane. Sherren, in conversation, told me of two that he had seen, but in neither case was the cancer at the site of the ulcer which was noted by him at operation. He was very definite on this point, and his figures and Patterson's certainly give him good grounds for his conservative attitude. I was so much impressed with Sherren's advice that I dropped the resection of gastric ulcers and performed only gastro-enterostomy in this condition, and so far I have no cause to regret it.

If you believe that there is a risk of cancer developing in a greater percentage than 2%, then logically you will perform partial gastrectomy or, at the very least, local excision by knife or cautery. If you believe that the incidence of cancer as a subsequent development is 2% or lower, then the only question to be decided is whether partial gastrectomy gives better results than simple anastomosis. In doing so, you must estimate the difference in the mortality rate in these two operations, and keep in mind that the biggest single factor making for success is adequate drainage of the resected or unmutilated stomach. In the master hands of Moynihan gastrectomy has been performed for years with the wonderfully low mortality rate of 1·6%. Most of the reports I have had access to vary from 6% to 25%.

I have a strong leaning to the opinion that gastro-enterostomy, with appropriate search beforehand for septic foci, proper pre-operative and post-operative treatment and proper attention to abdominal conditions, such as those of gall bladder, appendix and particularly a right-sided ptosis, is, in the hands of the majority of surgeons, likely to give the best results.

We are not yet in a position to state dogmatically the reason why these peptic ulcers fail to heal, and until we acquire that knowledge we cannot state what is absolutely the correct treatment. We must look to the combined efforts of the biochemist, bacteriologist, radiologist, physician and surgeon for advances sufficient to cover the deficiencies of our knowledge so that we may permanently relieve those poor sufferers who find "no shine in sun; no salt in sea."

SUMMARY.

1. Anatomical conditions and physiological disturbances bearing on the subject of peptic ulcer are briefly discussed.

2. Reference is made to the part played by associated abdominal conditions in the ætiology of peptic ulcer. Stress is laid on right-sided

ptosis and its significance in the peptic ulcer problem. Disturbance of the sympathetic nervous system is discussed in its relationship to ptosis.

3. Reference is made to infection by the lymph stream from the terminal portion of the ileum, the appendix and the caecum.

4. The surgical treatment of duodenal and gastric ulcer is discussed. Emphasis is laid on the importance of the search for septic foci and on the value of a properly performed gastro-enterostomy.

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NOTES ON TREATMENT OF FRACTURES IN THE LOWER LIMB, WITH SPECIAL REFERENCE TO BONY TRACTION.

By James M. Buchanan,
Melbourne.

In the treatment of fractures, the aim of the surgeon should be towards complete anatomical replacement. The Pensions Committee, set up in England after the war, found that the better the anatomical result, the better was the functional result, although there were some cases in which function seemed to be satisfactory when the anatomical replacement left much to be desired.

It is now generally accepted that extension in some form or another is essential in the treatment of fractures, particularly in the lower limb. Manipulation, under anæsthesia if need be, after adequate extension has been applied, will reduce the majority of fractures and produce a satisfactory position for immobilization. The reduction should then be maintained by continued extension until such time as sufficient callus has formed to knit the fragments together. The production of callus should be observed in X rays taken with a portable machine. The time of immobilization is therefore dependent upon the rate at which the individual produces new bone.

When the extension is finally removed and the patient allowed up, a walking caliper splint should always be used, in order to prevent bending of the soft callus which will inevitably occur if no form of supporting apparatus is fitted for the transmission of the body weight. The caliper splint is worn for eight to twelve weeks and gradually left off as the callus becomes solid. This method is preferable to the use of crutches in this stage, unless the patient is extremely conscientious in the matter of bearing weight through the injured limb.

Fractures without displacement or simple cracks in the bone need little or no extension and fixation by a moulded splint of plaster of Paris is usually sufficient.

With regard to the commoner methods of obtaining extension, it may be said at once that no pull can be taken from the soft parts in any way comparable with that obtained by means of bony traction. For example, extension by means of the Sinclair footpiece, glued to the foot by the flannel strips and triangles, must be strictly limited to 3·1 kilograms

(seven pounds). Any weight beyond this causes blistering of the skin, and in some cases in which the dorsum of the foot has been glued over (a technical error in the application), extensive sloughing has taken place. This result has nothing to do with the mode of application of the glue and strips. It is purely the result of undue weight being applied in unsuitable fashion.

Again, extension by long strips of gauze glued to the skin up each side of the limb or by adhesive plaster has the same disadvantage. Although a larger area of skin is being utilized, thus enabling the use of a slightly greater weight, any weight over 4·5 kilograms (ten pounds) will not be borne for any length of time without slipping of the adhesive or extensive blistering of the skin. Once this blistering has occurred and the extension has given way, the problem of obtaining further extension points or even of simple maintenance of the reduction becomes difficult. Of course, it may be that replacement of the fracture is easy, and in such a case 3·6 to 4·5 kilograms (eight to ten pounds) extension applied to the skin by gauze and glue or adhesive plaster may be all that is required to maintain the position whilst consolidation is occurring. Even so, the extension usually loosens owing to the desquamative action of the skin and requires to be replaced afresh about once a fortnight in young adults. In elderly people, as with intracapsular fractures of the femoral neck, such an extension may be satisfactory for a month or more at a time.

Hence, in treating severe fractures in which a high degree of extension is required, it has been found very satisfactory to take the pull from the bone itself. Traction up to 13·5 kilograms (thirty pounds) or more can be taken with certainty without pain to the patient and without any distressing skin sequelæ. Under such an extension, a large number of fractures may be reduced by manipulation. Should the reduction not be satisfactory, operative measures are rendered considerably easier if the limb has been subjected to an adequate extension for the preceding few days.

VALUE OF BONY TRACTION.

The mechanical principle that traction by means of rigid skeletal structures must of necessity be more efficient than any method which utilizes soft parts only, will be generally accepted.

The practical application of this principle has undoubtedly been delayed in the past by the feeling that the methods available violated in some respects present-day aseptic surgical desiderata. Consider, for example, the extraction of the Steinmann pin after the apparatus has been used for extension for some time: whatever means of purification of the exposed ends are adopted, a potentially infected piece of metal is drawn through a canal in the bone with a risk of sepsis and development of a chronic sinus in the bone.

Again, the ordinary types of caliper with points set into the bone at right angles, tend to become loose from osteoporosis after extension

has been maintained for some time. This leads to an inflammatory process, aseptic at first, upon which pyogenic infection not infrequently becomes implanted.[1] Thus the fear of osteomyelitis and chronic bony sinuses has swayed many surgeons against these methods. Hence a method of application of bony traction which eliminates these objections and at the same time provides a positive painless extension, has strong claims to an extended trial. Two such methods will be briefly described.

Screws in the Tibia.

Many ingenious methods have been devised for obtaining traction from the tibia. One of the simplest as well as one of the most effective is to introduce a pair of screws into the bone below the fracture and take the pull from them by means of sterile tapes. This method finds its most useful application in fractured femur (in any position) and high fractures of the tibia.

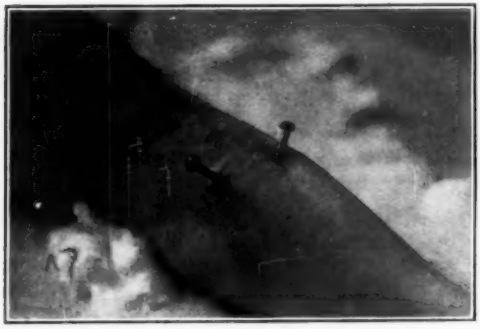

FIGURE I. Showing screws inserted into the tibia.

The technique, though simple, demands the highest aseptic precautions. The operation is performed with the leg supported by flannel slings, already in the Thomas knee splint. The area of skin through which it is proposed to operate is prepared beforehand by shaving, mechanical cleansing with ethereal soap, methylated ether and final application of 3% spirituous solution of picric acid. This picric acid solution was selected, after a thorough trial of the antiseptics used in war surgery, as being eminently reliable in this type of work.

The area of operation being isolated by sterile towels, stab wounds are made down to the subcutaneous and external surfaces of the tibia. A number 6 drill is passed down to the bone and bored through the compact bone and medulla to the opposite side. It is essential that the drill holes should be directed downwards, as there is a great tendency for the screw to loosen by osteoporosis, if the bone is bored at right angles to its surface. The drill is then removed, and a "three-sixteenth" screw 7·5 centimetres (three inches) long follows the track and bites on the compact bone at the other side of the medullary cavity. One screw on the subcutaneous surface and one on the external surface rather below the first one give an entirely adequate hold on the bone (see Figure I). About 2·5 centimetres (one inch) of each screw are visible above the skin after insertion, and the pull is then taken by looping sterile vaselined tapes round the heads of the screws. The vaseline is necessary to prevent the tapes being destroyed by

[1] The caliper devised by Corlette in which the points grip the bone from above downwards, has not this disadvantage.

constant exposure to picric acid. The whole of the projecting portions of the screws and the operation area are then completely enveloped in picric acid gauze and sterile bandages. Finally the long ends of the extension tapes are tied to the end of the Thomas splint or the foot prop, if used. After return of the patient to bed, the splint is suspended by four cords and a suitable weight (5·2 to 9·0 kilograms or twelve to twenty pounds) is applied to the end of the splint by cord and pulley. The foot of the bed is raised 20 to 30 centimetres (eight to twelve inches) by blocks and the weight of the patient is in large measure the counter-extending force.

This operation may be performed under local anæsthesia, though a general anæsthetic is often desirable if extensive manipulation is to be undertaken at the same sitting. Screws, thus inserted into the bone, give

FIGURE IIA. Showing effect of extension upon an impacted fracture (antero-posterior view). The positon of the fragments is seen under gauze and the glue extension of 4·5 kilograms (ten pounds).

FIGURE IIB. Showing effect of extension upon an impacted fracture. This figure is a lateral view of the fracture shown in Figure IIA.

a firm, painless grip upon the skeletal structure of the limb. If necessary, they may be retained in the tibia for six to nine months, as in cases of femoral non-union, without discomfort or untoward symptoms. They are removed by simply unscrewing (no anæsthesia being required), and the small, granulating wound heals readily within fourteen to twenty-one days. No unfortunate occurrence, such as osteomyelitis of the tibia, has been noted to follow their use. Undoubtedly, however, 2% to 3% do develop a minor degree of infection at the site of passage through the skin without apparently affecting the subjacent bone. The infecting organism is commonly the *Staphylococcus albus*. The cause of this infection is usually found to be some oversight in the technique of

application. Thus the skin preparation may not have been entirely adequate or, more important, the screws have been inserted at right angles to the bone surface. If this is so, the screw soon loosens and these infections are almost always noted around loose screws.

Provided the screws are inserted correctly, they remain firm and perfectly clean for months at a time.

Figure II shows the effect of bony traction by screws in the case of an impacted fracture of the upper third of the tibia. This patient had been treated by manipulation under anæsthesia, with traction by means of gauze and glue attached to the skin along the whole length of

FIGURE IIc. Showing effect of extension upon an impacted fracture (antero-posterior view). The same limb as depicted in Figures IIA and IIB after extension by screws with nine kilograms (twenty pounds).

FIGURE IID. Showing effect of extension upon an impacted fracture. This is a lateral view of the limb depicted in Figure IIc.

the leg below the knee. It will be seen in Figure IIA that little effect has been produced. After screws had been inserted into the tibia and an extension of 9·0 kilograms (twenty pounds) maintained for four days, the fragments were disimpacted and the posterior displacement of the upper fragment completely reduced. There was at this stage some degree of over-extension. All that now remained was to lessen the weight to 4·5 kilograms (ten pounds) and allow the bone to consolidate. This took place in some six weeks, and the patient then walked

in a caliper splint for eight weeks until complete consolidation had occurred.

Fractures About the Hip Joint.

In fractures about the hip joint, especially the extracapsular variety occurring in muscular adults, screw traction from the tibia has been found very satisfactory. Figure III shows the results in such a fracture. The usual weight applied is 5·2 to 6·75 kilograms (twelve to fifteen pounds), the foot of the bed being raised 20 to 25 centimetres (eight to

FIGURE IIIA. Extracapsular fracture of the neck of the femur. Before screw extension.

FIGURE IIIB. Extracapsular fracture of the neck of the femur. During screw extension.

ten inches) to provide counter-extension. The absence of the *coxa vara* deformity which often follows these fractures on consolidation, is noticeable.

Figure IV shows the X ray photographs in the case of a central intra-pelvic dislocation of the hip joint. Manipulation and high traction by screws in the tibia produced a satisfactory reduction. It should be noted that no fracture involving the pelvic girdle will be benefited by a walking caliper, because the caliper takes the thrust from the *tuber ischii*. Prolongation of the period of recumbency or careful mobilization with crutches is therefore essential in these cases.

There appears to be little cause to fear weakening of the ligaments of the knee joint, although there is, to be sure, some stiffness when the extension is finally removed. The tone of the muscles having been

maintained, however, by massage during the period of extension, the movement in the knee rapidly improves and, as a rule, is complete by the time the patient discards his walking caliper splint and walks without mechanical aid.

Fractures of the Shaft of the Femur.

With regard to fractures of the femoral shaft, the initial treatment should be adequate extension by means of screws in the tibia and manipulation in which an endeavour is made to correct the displacements seen in the X ray picture. The most obstinate of these displacements is undoubtedly the posterior position of the upper end of the lower fragment. This must be corrected, whatever the means adopted, otherwise some degree, more marked in some cases than in others, of *genu recurvatum* is the result—a most crippling deformity

FIGURE IIIc. Extracapsular fracture of the neck of the femur. Showing final result after screw extension.

The result of the recognition of this fact is that surgeons generally are relying more and more on early open operation in cases of fractures of the shaft of the femur with much displacement. During the actual operation, the ease with which the fragments are reduced and brought into perfect apposition is determined absolutely by the amount of extension of the limb during these manipulations. In cases where no extension is used, very great difficulty may be encountered in bringing the fractured ends into apposition. Various mechanical devices can be used as attachments to an operating table for obtaining extension during operation.

When screws have already been inserted into the tibia for primary extension, the mechanical extension is ready for use. All that is necessary is to hook the foot prop of the Thomas splint to the end of the table and tilt the head end downwards. The amount of extension is regulated by the amount of tilting: the splint and lower fragment (held by the tapes and screws) remain fixed, while the entire body weight of the patient dropping away from the splint, exerts an excellent traction upon the upper fragment. In this simple way the extension required for operative reduction can be varied at will.

The operation is performed with the limb in the Thomas splint, screws having been inserted into the tibia beforehand. The area of operation having been sterilized and the supporting bands being removed, the splint is eliminated from the operative field by a suitable arrangement of sterile sheets and towels. The approach to the femur should be by antero-lateral incision which separates the *crureus* from the *vastus lateralis*; little or no bleeding is then encountered. The fractured bone ends should be cleared thoroughly with long sharp raspatories to remove all soft tissue from between the fragments. Manipulation with two pairs of lion forceps grasping the bone close to the site of fracture then brings the fragments into good position, this step being very much aided by the extension manœuvre alluded to above.

FIGURE IVa. Showing intrapelvic dislocation at the hip joint before extension and manipulation.

When the position is satisfactory, a bone clamp is placed round the site of fracture and locked. Silver wires or a Lane's plate can then be applied at leisure. The extension is maintained while the mechanical fixation is effected. A sterilized flannel band is then placed under the limb at the site of fracture and clipped to the splint, thus preventing any tendency to posterior displacement. The wound is closed with interrupted silkworm gut sutures. When the patient returns to the ward a weight of 3·6 to 4·5 kilograms (eight to ten pounds) should always be applied, as the mechanical fixation is insufficient without the extension.

Silver wire (sixteen to eighteen gauge), when used to encircle the bone, may be retained *in situ* indefinitely. Lane's plates, on the other

hand, should be removed as soon as sufficient callus is revealed by X ray examination (usually about six weeks). This precaution is necessary because metastatic infection and abscess formation have been observed to develop round these plates four and five years after symptomless recovery. As the patient may by that time be out of reach of surgical aid, it is a wise step to forestall any such mishap and remove plates as soon as their mechanical function has ceased.

Figures V and VI illustrate methods of dealing with fracture of the femoral shaft by the procedures just mentioned.

FIGURE IVB. Showing intrapelvic dislocation at the hip joint after extension and manipulation.

Fractured Tibia and Fibula.

Many fractures involving the tibia and fibula can be satisfactorily reduced and retained in good position by extension and manipulation.

There are, however, certain fractures in which open operation is essential. Figure VII illustrates such a case. Here the violence was applied directly across the tibia and it might appear from the skiagram that the only procedure necessary to reduce this fracture is extension and lateral pressure. This is not the case, however. Such a fracture at open operation requires a rotatory movement of almost 180° to disentangle the muscle in which its fragments are invariably caught.

It is to this type of fracture that the lower half of the tibia owes its reputation for non-union. Open reduction and fixation, either by impacting the ends of the bone or by mechanical means, such as plates, give adequate results in such cases.

The long spiral fracture of the tibia near the ankle in an adult is also benefited by open operation (Figure VIII). Wiring the bone in these cases gives a perfect position which is not always obtained by simple extension methods. However, as the results from non-operative

FIGURE Va. Showing spiral fracture of the shaft of the femur.

FIGURE Vb. Showing spiral fracture of the shaft of the femur after wiring.

treatment are usually good, judgement must be used to select those cases in which operation is indicated.

The technique of operation in this situation is the same as used for the femoral shaft, that is, the operation is performed with the limb in the splint, isolated by suitable arrangement of towels. The fracture is exposed by incision over the subcutaneous surface of the tibia. Reduction is secured under extension obtained by a temporary clove hitch round the ankle and serrated wooden foot-

piece, and fixation is carried out by plating, wiring or simple impaction of the fragments without additional mechanical support. An extension of 3·1 kilograms (seven pounds) is maintained from the footpiece after operation.

FIGURE VIA. Showing transverse picture of the femur.

FIGURE VIB. Showing transverse fracture of the femur after plating.

Heel Stirrup.

The heel stirrup was designed by Major Sinclair to eliminate a disability common to other forms of traction over the *os calcis*, namely, the necessity of drawing a potentially infected piece of metal or wire through a track in the soft tissues with risk of infection and a persisting sinus. The points of the instrument (seen in Figure IX) are sterile when inserted, and, by separation on withdrawal, only the parts which have

been buried in the tissues during the period of traction pass outwards. There is thus no risk of carrying any infection inwards during withdrawal, as is always possible, for example, with Steinmann pins.

The instrument consists of two pieces of metal hinged at the base and curved inwards at their upper ends. Notches in the basal bars agree identically when the points of the instrument are in contact; this assures firm hold and gives a definite point to which the traction tapes may be attached.

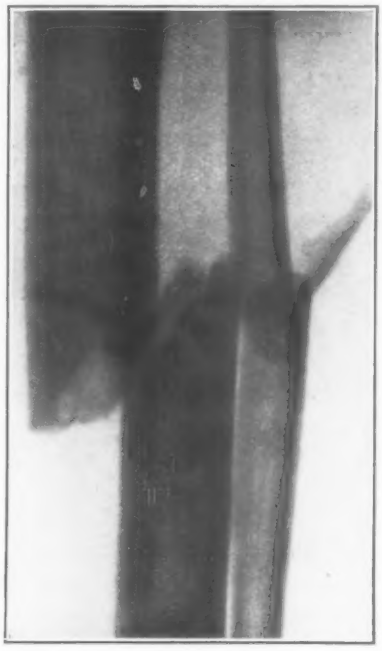

FIGURE VIIA. Showing transverse fracture of the tibia which usually entangles muscle tissue between the fragments.

FIGURE VIIB. Showing transverse fracture of the tibia (seen in Figure VIIA) after plating.

FRACTURES ABOUT THE ANKLE JOINT.

The method to be adopted in dealing with fractures round the ankle joint (so-called Pott's fracture) depends to a large extent upon a close scrutiny of X ray pictures taken in two planes showing the damage and displacements.

In cases without gross deformity, the treatment consists in immobilization of the ankle in good position, that is, at right angles and slight inversion, until such time as the swelling produced by the injury has

subsided. This usually takes ten to fourteen days. For this purpose the Thomas splint with the serrated wooden footpiece, or a back splint with footpiece, may be used. At the end of this time the limb should be fitted with an accurately moulded plaster cast extending from the toes to just below the knee. Daily massage is instituted, the cast being removed

FIGURE VIIIA. Showing spiral fracture of the tibia (and fibula), usually high up near the neck of the bone.

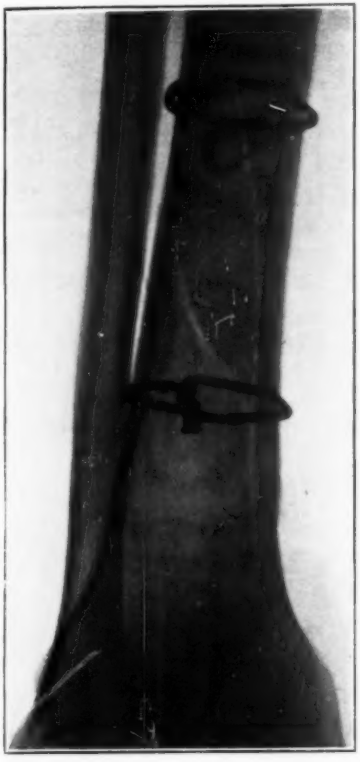

FIGURE VIIIB. Showing spiral fracture of the tibia after wiring.

for the purpose. The cast should be retained for about a month and the patient is then allowed to walk in a pair of boots, the sole of the boot on the injured side being wedged upwards about six millimetres (a quarter of an inch).

If, however, there is gross displacement, mobile extension produces better results. The extension in this situation is obtained by means of Sinclair's stirrup, which is seen in Figure IX. By means of two stab

wounds, one on either side, immediately in front of the *tendo Achillis* and above the *os calcis*, the two curved traction bars of the stirrup are brought into contact over the *os calcis*. The bars are locked in position by looped tapes taking up the notches in the base of the stirrup. These tapes are firmly tied to the foot prop at the end of the Thomas splint. The upper part of the foot is controlled by a serrated wooden footpiece in the ordinary way. If the footpiece is very wide, it may be necessary to cut away some of its lower notches in order to allow it to pass down through the stirrup. Manipulation is undertaken as soon as the stirrup is in position and extension thus obtained.

Should the position not be satisfactory, further manipulation of the fragments is always possible under gas anæsthesia without moving the patient from his extension frame. Satisfactory reposition is the rule in cases treated on these lines.

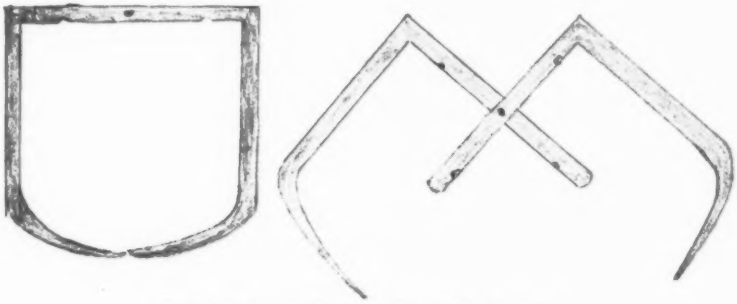

FIGURE IX. Showing the Sinclair heel stirrup.

After maintenance of the reduced position by continuous extension for about six weeks, depending upon the severity of the original displacement, these cases may be mobilized after being fitted with an ankle support. This commonly takes the form of a six millimetre (one-quarter inch) inner wedge on the boot and an external iron with internal "T" strap to prevent *valgus* deformity. The iron and boot should be worn in bad cases for four to six months.

Figure X shows the stages in treatment of a Dupuytren fracture. The efficiency of the extension in this case is shown by the marked separation of the articular surfaces of the tibia and the *talus* while the fracture was under traction of 6·75 kilograms (fifteen pounds). That this separation causes no permanent disability is seen by the final pictures.

SUMMARY.

Various types of fracture of the lower limb which are advantageously treated by bony traction are mentioned. Bony traction is not advocated as a routine treatment for all fractures, as many can be satisfactorily

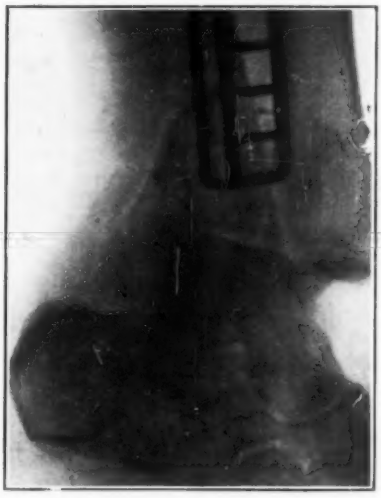

FIGURE XA. Showing fracture dislocation at the ankle joint.

FIGURE XB. Showing fracture dislocation at the ankle joint reduced under extension by the stirrup.

FIGURE XC. Showing final result in fracture dislocation at the ankle joint. Antero-posterior view.

FIGURE XD. Showing final result in fracture dislocation at the ankle joint. Lateral view.

treated by simpler methods. However, it is suggested that bony traction should be used wherever adequate extension cannot be obtained by pull from superficial structures.

Gross displacements which do not respond to adequate extension and manipulation should be submitted to open correction, particularly those involving the shaft of the femur.

Certain methods of application of bony traction are briefly described.

ACKNOWLEDGEMENT.

The aim of the foregoing paper has been to present, in concise form, methods observed whilst working in London with Major Meurice Sinclair, the originator of the serrated wooden footpiece, which is used in conjunction with the Thomas knee splint for fractures in the lower limb. The author wishes to acknowledge gratefully the unfailing help, encouragement and courtesy that he always enjoyed in his association with Major Sinclair.

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TREATMENT OF HALLUX VALGUS AND HALLUX RIGIDUS.

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In many cases of *hallux valgus* and in all cases of *hallux rigidus* deformity of the head of the first metatarsal bone and limitation of movement are the outstanding features. In such instances operations upon the soft parts alone are doomed to failure. When there is no deformity of the head of the metatarsal bone, adduction of the great

Figure I. Showing line of excision.

toe may be attributed to a condition of the first metatarsal bone known as *metatarsus primus varus.*[1] This will be again considered under the operative procedures.

The commonest effective surgical procedure in the treatment of *hallux valgus* and *hallux rigidus* is excision of the head of the metatarsal bone. This procedure leads to a great shortening of the great toe and is often followed by pain in the region of the sesamoid bones of the great toe or in the heads of the other metatarsal bones.

I have devised a new operative procedure in order to obviate these disadvantages, and have carried it out in nineteen instances.

THE OPERATION.

An incision is made along the medial side of the metatarso-phalangeal joint of the great toe, extending from about three centimetres in front of the joint to about six centimetres behind it (Figure I). The skin flaps are dissected up and retracted. An incision is then made

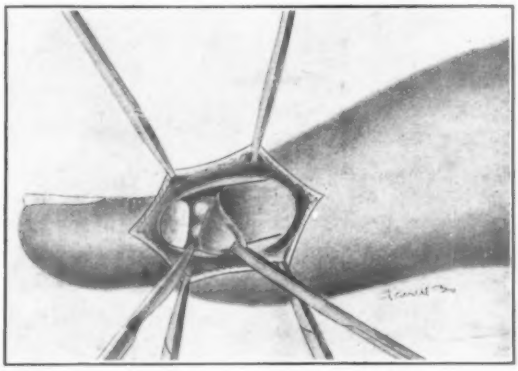

FIGURE II. Dissection of flap commencing.

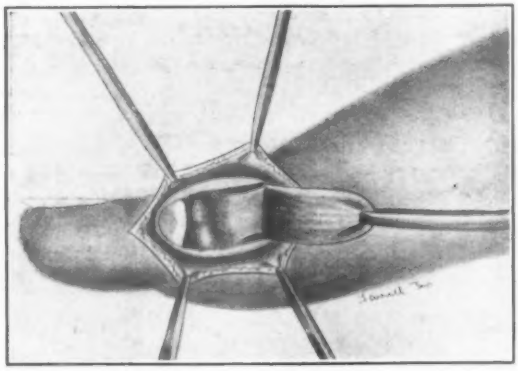

FIGURE III. Dissection of flap completed.

through the anterior part of the capsule of the joint (supero-inferiorly) as far forwards as possible. From each extremity of this incision the knife is carried backwards and a flap marked out which is then dissected posteriorly as far as the neck of the metatarsal bone (Figures II and III). The metatarso-phalangeal joint is thus opened. An osteotome is then used to cut off about one centimetre of bone from the anterior

end of the head of the metatarsal bone. This section only partially removes the articular surface of the head, and must be made in any case distal to the sesamoid bones. The head is then shaped with bone-cutting scissors and with a file to present a conical anterior extremity as shown in Figure IV. The flap (*a*) is then placed round this

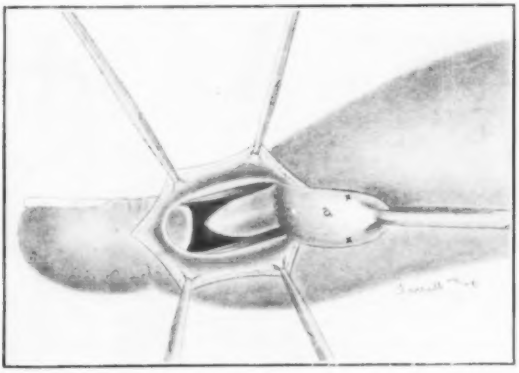

FIGURE IV. Showing reshaped head of metatarsal bone.

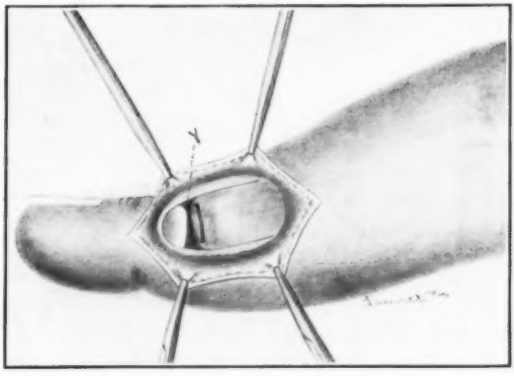

FIGURE V. Showing flap in position.

conical end and is stitched in place by means of fine catgut threaded on needles at both ends. One needle pierces the flap at the upper edge and the other passes through the lower edge and each is brought through the skin in the superior aspect of the first intermetatarsal space (Figure V). This procedure holds the flap in position. The wound is then closed with a single cutaneous stitch of fine catgut.

If *metatarsus primus varus* be present, a simple osteotomy just distal to the base of the metatarsal bone from without inwards will correct the deformity. The capsule may be incised on the lateral side if necessary. The wound is dressed and the foot is enclosed in a light plaster of Paris cast. The purpose of this cast is to support a wire extension piece shaped as shown in Figure VI.

A most important detail in this procedure is to apply toe nail extension to the great toe. This is done by drilling the toe nail with a straight needle, in the eye of which is threaded strong silk. For this purpose it is wise to leave the great toe nail long. The silk is attached

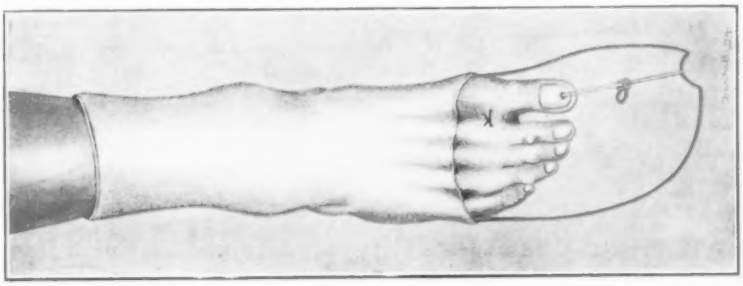

FIGURE VI. Showing extension and flap stitch in position on the dorsum of foot.

to a rubber band which is so adjusted as to keep apart the base of the first phalanx and the reshaped head of the metatarsal bone.

Care must be exercised that the tension applied is not too great, otherwise the toe nail may be dragged from its bed. The object of extension is to keep apart the components of the reformed joint until fibrous tissue has been formed in space (Y, Figure V). This occurs in from ten to fourteen days. Active and passive movements are encouraged while the extension is on, and may be commenced in two or three days after operation. The patient should be instructed to persevere with movement at the reformed joint to prevent the fibrous tissue between the bony components from contracting. The patient may walk in slippers at the end of ten days, but it is usually three or four weeks before shoes can be comfortably worn, on account of the swelling following the operation. There is no difficulty in getting full plantar flexion and dorsiflexion immediately after operation and these movements do not become restricted.

SURGERY IN DIABETICS.[1]

By JAMES R. BELL,
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DURING the past decade the outlook for the diabetic individual has entirely changed. Before the advent of insulin his lot was an unenviable one, being often that of a semi-invalid living in constant danger of some intercurrent illness or infection precipitating acidosis, coma and death. The discovery of insulin by Banting and his colleagues at Toronto has transformed the diabetic's life, and shorn the disease of many of its terrors. The diabetic child may now thrive at least as well as the non-diabetic; the diabetic woman may safely pass through pregnancy and be delivered of a healthy infant; the dreaded coma is now a penalty of neglect and ignorance, and should therefore be avoidable; while, not least of all, the diabetic may enjoy the benefits of surgery with very little, if any, greater risk than the non-diabetic.

During 1929 the operative mortality of diabetics at the Montreal General Hospital was 2·74% in 73 cases, while the total operative mortality was 2·41% in 5,750 cases. My own experience during the past eight years is in accord with these statistics. This remarkable achievement ranks high among the gifts of medicine to surgery, yet some surgeons are still unduly reluctant to operate upon a diabetic patient.

Before considering the problem of the surgical diabetic, may I remind you that glycosuria does not spell diabetes. In many cases blood sugar estimations are necessary to determine this question.

There are certain general principles in the management of the surgical diabetic, their main object being to control severe post-operative acidosis and to facilitate satisfactory healing of tissues. These principles are:

(1) Effective cooperation between physician and surgeon, assisted by the laboratory and dietetic departments. This is no mere platitude, but, as Joslin says, "the keynote of surgery in diabetes."

(2) The patient should be in a good state of general nutrition. For the diabetic this is always desirable, but especially so before an operation.

[1] Being the Physician's contribution to the discussion on diabetes at the annual meeting of the College of Surgeons of Australasia, Sydney, April, 1931.

(3) Abundant glycogen storage, which should not have been depleted by prolonged fasting or semi-starvation.

(4) Freedom from ketosis is desirable whenever time permits of its control. The *liquor ferri perchloridi* test of the urine is a reliable and simple index.

(5) The patient should not be dehydrated, as this predisposes to ketosis.

(6) The sugar content of the blood should not be unduly high. Although it has been urged that the blood sugar should be as nearly normal as possible, I do not consider this is essential in the pre-operative stage.

(7) Chloroform anæsthesia must always be avoided, and ether if possible. Ethylene and gas and oxygen are reasonably safe, while local anæsthesia is the ideal method.

Certain of these guiding principles may now be amplified in considering the pre-operative treatment of an average case. It is desirable that the patient spend three to four days in hospital before operation, but occasionally a longer period is necessary. Even a patient with mild diabetes should be given insulin at this stage as an additional safeguard against post-operative acidosis.

A diet is planned to consist mainly of readily assimilable high-grade carbohydrate foods, such as oatmeal gruel, cereals, fruits, bread and toast, with tea or coffee to which glucose may be added. In addition, meat, fish, eggs, vegetables, cream and butter are allowed sparingly. The aim is to provide at least 100 grammes of easily assimilated carbohydrate each day. Sufficient insulin must be given, about fifteen minutes before each meal, to keep the urine sugar-free, and so insure the storage of ample reserves of glycogen. About three to four hours before operation the patient is given half to one ounce of oatmeal, made into gruel, to which is added one to two teaspoonfuls of glucose. This is, of course, preceded by an appropriate dose of insulin.

Excessive purging is unwise during this time of preparation, and at least three pints of fluid should be given daily.

In cases of emergency, when time does not permit of this deliberate scheme, half a litre (about one pint) of a 10% glucose-saline solution, containing insulin, should be injected intravenously. The amount of insulin added depends upon the degree of hyperglycæmia present. If a blood sugar estimation is impracticable, one must be guided by the amount of glycosuria present. A suitable average dose would be 50 to 60 units of insulin. The glucose-insulin-saline solution must be injected slowly, and the injection completed during the course of the operation if necessary. It is essential that the glucose be chemically pure; in my experience no untoward effects have followed the use of British Drug Houses' anhydrous glucose.

Post-operative treatment is at least as important as the pre-operative, but the more efficiently the patient is prepared before operation, the less difficult and worrying will be the post-operative care.

Acidosis must be controlled at all costs, and this is best done by insuring adequate metabolism of carbohydrate. For this purpose large amounts of insulin may be necessary, while carbohydrate must be freely given as a safeguard against undue lowering of the blood sugar and to provide sufficient antiketogenic material. It matters little if glycosuria is present during the first day or two after operation, so long as ketonuria is controlled.

Immediately after operation 300 cubic centimetres (one-half pint) of 10% glucose-saline solution should be given by rectum. Opinions differ as to whether insulin should be added to the glucose-saline solution or injected subcutaneously. It is undoubtedly absorbed from the bowel, but the effect produced on the blood sugar content is much less than that following subcutaneous injection. Should the glucose-saline solution be not retained, or the rate of absorption be too slow, insulin given subcutaneously may cause hypoglycæmia. However, this is unlikely if moderate doses are given, as a considerable rise of blood sugar occurs after all operations requiring a general anæsthetic. In most cases the addition of 50 units of insulin to the 300 cubic centimetres of glucose-saline solution seems to be effective, supplemented by smaller subcutaneous doses if necessary. The rectal injections should be repeated every three hours, if practicable, until the patient can take light nourishment by mouth. Occasionally the glucose-insulin-saline solution must be given intravenously, and this may be done three times in twenty-four hours.

During the first few days after operation blood sugar estimations are not, as a rule, essential. One may safely rely upon frequent testing of the urine, specimens being obtained at intervals of three to four hours, if necessary by catheterization. Blood sugar estimations are desirable in certain difficult cases as a guide to insulin dosage and where the healing power of the tissues is poor, as it is then essential to maintain a low level of blood sugar.

Vomiting must be prevented if possible, and controlled without delay if it occurs, for excessive post-operative vomiting rapidly induces acidosis. A "vicious circle" is set up, whereby the continued vomiting increases the acidosis, and the acidosis still further aggravates the vomiting.

As soon as the patient can retain food, cereals, toast or bread and fruit juices are given with sufficient insulin to control glycosuria. Frequent moderate doses are preferable to occasional large doses at this stage, to avoid hypoglycæmia. Plenty of fluids are essential. Appropriate additions are made to the dietary during convalescence, and the carbohydrate and insulin decreased until the stage of maintenance diet, with or without insulin, is reached. When the blood sugar cannot be kept at an approximately normal level by reasonable dietary measures alone, insulin should be continued for at least a month or so.

From the vast range of surgical pathology, may I now briefly refer to certain conditions of outstanding interest.

Gall Bladder Disease.—Glycosuria is frequently associated with cholelithiasis and cholecystitis, while not a few cases of diabetes are initiated by long standing gall bladder disease. It is always desirable to remove a pathological gall bladder in a diabetic, as it may, and often does, lead to an improvement in the diabetic condition.

Thyroid Disease.—The combination of hyperthyreoidism and diabetes is a serious one, for the increased metabolic rate associated with hyperthyreoidism demands a greater calorie intake—not infrequently double the usual requirement. Now over-eating is harmful to the diabetic condition, and still more so is an increased metabolic rate. The patient's carbohydrate tolerance is usually improved by thyreoidectomy, sometimes to a remarkable extent. Joslin says: "Surgery in the hyperthyroid diabetic is the best sort of a game, because it is exciting and if you know and follow all the rules you are pretty sure to win." It certainly may tax to the limit the cooperative efforts to which I have already referred.

Carbuncles and Other Pyogenic Infections.—The well treated diabetic is no more susceptible to carbuncles and other pyogenic infections than the non-diabetic individual. When they occur through neglect or inefficient treatment, they may be most intractable. Carbuncles are often fatal, and surgical intervention should not be delayed, for the toxæmic effects are usually severe and the patient may die despite complete control of hyperglycæmia and ketosis. In all infective conditions larger doses of insulin are necessary, for infection in some way appreciably lessens the insulin effect. Reliance must not be placed upon insulin alone, and prompt surgical treatment of the infection is essential.

Ulcers: Superficial and Penetrating.—In all conditions of severe tissue damage it is futile merely to control glycosuria and allow excess of sugar to remain in the blood. The blood sugar should be kept at a slightly subnormal level, with the patient verging on hypoglycæmia. By this procedure, which I am convinced is the *sine qua non* of effective treatment, the rate of healing is sometimes remarkable, almost exceeding that of similar conditions in non-diabetic individuals. The callous penetrating ulcer is a problem in itself, and surgical intervention may be necessary.

Diabetic Gangrene.—Largely through the work of Buerger, the view has been widely held that the arterial changes in diabetic gangrene are the same as in arteriosclerotic gangrene. The correctness of this view, however, has been recently disputed.

Gangrene is now the gravest surgical complication of diabetes. Whereas at the Montreal General Hospital the percentage of deaths from all causes in diabetes has fallen since the advent of insulin from 12% to 3·8%, that from gangrene has increased from 2·8% to 4·6%.

The incidence of gangrene in diabetics is about 3%, and depends on age rather than the duration of the diabetes. According to the

statistics of Joslin and others the average age is about 60 years, while one-quarter of all diabetics past the age of 70 years develop gangrene. It is an interesting fact that the incidence of syphilis is about seven times as great in diabetics with gangrene as in those with no gangrene.

Efficient control of the diabetic condition greatly lessens the tendency to gangrene, and Joslin has emphasized the importance of scrupulous care of the feet (for the lower extremities are most often affected).

In the milder cases, where the gangrene is dry and localized, non-operative treatment may be effective. It is essential that a slightly sub-normal blood sugar level be maintained, while Buerger's exercises, alternate heat and cold, radiotherapy and periarterial sympathectomy may be of value in selected cases.

When the gangrene is extensive, and eventual amputation inevitable, there may be no great operative urgency. In such cases surgical operation is best postponed until appropriate medical treatment has improved the patient's general condition.

The septic, moist type of gangrene, with toxic symptoms, unquestionably demands immediate amputation. The operative risk is great, but is to some extent lessened by intravenous glucose-insulin-saline therapy.

In conclusion, may I briefly refer to the condition known as "diabetic acute abdomen." It is an occasional feature of marked ketosis or incipient coma, and may closely simulate an acute inflammatory condition of the abdomen. The differential diagnosis is difficult, and may be impossible. In the three cases that I have seen, the absence of definite localizing signs and pyrexia fortunately proved helpful in arriving at a correct diagnosis. Nevertheless, if any reasonable doubt exists, an exploratory laparotomy is justified.

Case Reports.

INTRABILIARY RUPTURE OF AN HEPATIC HYDATID CYST.

By B. T. ZWAR,
Honorary Surgeon, Melbourne Hospital.

THE phenomenon of intrabiliary rupture of a hydatid cyst of the liver is one long recognized clinically, but in the living it has been difficult to demonstrate the rupture or communication. Dew in his book on hydatid disease expresses the conclusion: "That some degree of intrabiliary rupture is the commonest of all complications of hepatic cysts." In Australia it is usual to take into consideration hydatid disease of the liver as a possible cause of biliary colic associated with cholangitis and jaundice. The intrabiliary rupture of an hepatic hydatid and the passage of small daughter cysts, bits of membrane or small particles of *débris* may give rise to cholangitis and biliary colic indistinguishable from those caused by biliary calculi. The following case is one of this nature; it is recorded because the X ray investigation carried out during the course of the post-operative treatment demonstrated the communication between the cyst cavity and the larger biliary passages.

The case is that of a farmer, aged fifty-seven years, born and brought up in a country district of Victoria, who on November 13, 1929, presented himself for examination with a mass in the upper part of the abdomen. The mass occupied the whole of the epigastric area and both right and left upper abdominal quadrants to a hand's breadth below the costal margins. It was most prominent in the upper left quadrant.

The patient stated that he had noticed a lump in the left side "under the ribs" about twenty-seven years previously. It had been noticed shortly after an accident—a fall off a bicycle—and its appearance had been associated with severe abdominal pain. At that time he had sought medical advice and had been told that the swelling was due to a traumatic aneurysm of the aorta, and he had been ordered absolute rest in bed. At the end of three months in bed he had had an attack of abdominal pain associated with collapse and urticaria. Exploratory needling of the right hepatic area had then been carried out, but, although a quantity of clear fluid had been aspirated, no surgical treatment had been advised. The mass on the left side had not undergone any apparent change and the patient had been assured again that this was due to an aneurysm of the aorta and had been advised to regulate his mode of life from this point of view. The patient stated that he had preferred "to take the risk" and that he had taken "little heed" of the advice. Recurring attacks of abdominal pain, discomfort after meals, increasing constipation and loss of weight had induced him now to seek medical aid.

At the time of examination he complained of attacks of severe right-sided abdominal pain which radiated from the right hypochondrium towards the umbilicus and towards the region of the right shoulder blade. At times the pain had been so severe as to double him up. Of late he had been much troubled with

obstinate constipation. He complained that even a small meal would be followed by a feeling of epigastric fullness and discomfort. His weight which two months ago had been 80 kilograms (twelve stone ten pounds), was now 75·6 kilograms (twelve stone).

Examination disclosed a mass occupying the upper part of the abdomen as already described. Palpation of the mass gave a feeling of great tenseness, especially over the epigastric and left portions; pulsation was readily palpable in the mid-line portion. Percussion gave a dull note over the whole of the mass; on the right side the subcostal dulness merged into that of the normal liver dulness, which was also increased in an upward direction. No thrill could be elicited. The temperature was 36·7° C. (98° F.), the pulse rate 60; the systolic blood pressure was 120 and the diastolic pressure 80 millimetres of mercury. Examination of the urinary, respiratory and cardiac systems did not disclose anything abnormal.

The complement fixation test for hydatid, carried out at the Walter and Eliza Hall Institute, gave a triple positive reaction by both the warm and the ice-box methods. The Casoni test gave an immediate positive reaction. X ray examination by Douglas Thomas showed that the right diaphragm was somewhat raised and had very poor movement. This was secondary to hepatomegaly. In the films the enlarged liver showed two suggestions of cyst outlines, one in the right lobe and one in the left. That in the left was quite definite; the details in the right were not so clear; they suggested an old cyst high up in the right side of the liver fairly centrally situated.

The patient was submitted to operation on November 18, 1929. The abdomen was opened by a left upper paramedian incision, the anterior sheath of the left rectus being divided some distance from the mid-line and the left rectus was retracted laterally. Immediately upon division of the posterior sheath and peritoneum the tense glistening white tumour mass arising from the left lobe of the liver came into view. This mass was carefully packed off from the surrounding structures, including the parieties. Twenty cubic centimetres of clear hydatid fluid were then drawn off through a serum syringe and replaced by an equal quantity of 5% solution of formalin. After a brief interval the adventitia, which ranged in thickness from three to six millimetres (one-eighth to one-quarter of an inch), was incised and about 2·5 litres (five pints) of hydatid fluid and daughter cysts were removed by means of a spoon and suction pump. The contents were clear and no bile was noted until near the end of the emptying, when a liberal admixture of bile made its appearance. It was found that the intrahepatic portion of the cavity extended over to the right. During the emptying several quantities of 5% solution of formalin were introduced and when the cavity had been emptied as completely as seemed possible, it was swabbed out with gauze moist out of 5% formalin solution and packed with sterile gauze. Considerable time was then spent in resecting as much as possible of the extrahepatic portion of the adventitia. The operation was completed by removing the packing, again swabbing the cavity with gauze moist out of formalin solution, and securely suturing its opening around a drain tube 2·5 centimetres (one inch) in diameter. A catgut suture anchored the lower part of the opening to the peritoneum and posterior sheath. The parietal wound was closed in layers. The progress of the patient's convalescence was interrupted by recurring attacks which presented the clinical picture of infective cholangitis. The post-operative treatment consisted of daily irrigations; generally with 1% formalin solution. Hexamine, 0·3 gramme (five grains), was given night and morning and during the attacks of cholangitis this was supplemented with intravenous injection of two cubic centimetres of a 2% solution of "Euflavine" on alternative days. Drainage was assisted by decubitus in the prone position for short intervals each day.

For the first four days the discharge was blood stained and copious, and contained numerous small daughter cysts, fragments of hydatid membrane and *débris*; the temperature remained subnormal. On the fifth day the patient had a mild rigor, the temperature rose to 38·5° C. (101·4° F.), the discharge diminished

in amount and became bile stained and mucoid in character. A swinging type of temperature with a maximum variation between 36·7° and 38·9° C. (98° and 102° F.) continued for seven days. During this period the patient complained of pain in the gall bladder region and of tenderness on pressure over this area, he became slightly jaundiced and the stools tended to be clay coloured.

With the return to a normal temperature the discharge became free of bile and mucus, whilst the pain and tenderness disappeared and there was progressive improvement in the patient's general condition. Febrile attacks similar to the

above occurred at the end of the eighth week, at the beginning of the eleventh and during th thirteenth and fifteenth week. On an average these attacks lasted for five to six days; they were all characterized by the appearance of bile and thick ropy mucus in the discharge and presented the clinical picture of an infective cholangitis. In the intervals between the attacks the temperature *et cetera* ran a normal course.

At the end of the last attack during the fifteenth week it was decided to attempt to trace the connexion between the hydatid cyst cavity and the biliary apparatus. It was hoped that the X ray examination might supply information uueful in effecting more efficient drainage of the biliary passages and the cyst cavity. The outlet of the cavity by this time had diminished to a tract admitting a number 14 urethral catheter and through a tightly fitting tube of that size

a quantity of a 15% solution of sodium iodide was introduced into the cavity by means of a funnel, and the accompanying X ray film, taken by Dr. Thomas, was obtained. It demonstrates the communication of the cavity with the biliary passages and shows distension with the iodide solution of the hepatic, cystic, common ducts and gall bladder as well as the flow of the solution into the duodenum.

The procedure was not followed by any untoward effect; on the contrary, the patient insisted that he felt benefited by it. Whether as a matter of coincidence, or on account of the injection, his subsequent convalescence was not interrupted by any further attacks of cholangitis. It is possible that the injection may have flushed out of the biliary passages infective *débris* which these harboured. Under the circumstances the question of choledochostomy which had presented itself, did not require further consideration.

A METHOD OF TREATING WITH RADIUM CARCINOMA IN REGION OF SOFT PALATE.

By E. GORDON ANDERSON,
Surgeon, Wellington Hospital.

THE treatment of neoplasms with radium has come very much into vogue within the last few years. The mode of application recommended is so varied, the strength of applications so different and the screenage thickness so erratic that the novice is bewildered when he endeavours to work out a plan based upon the experience of the many published methods.

In submitting the accompanying description I am keenly aware that I have only one case to describe. The peculiar difficulties of the location, I believe, justify my publishing the way in which my colleague and I got over our difficulty. I trust that this description will help another to handle the same problem.

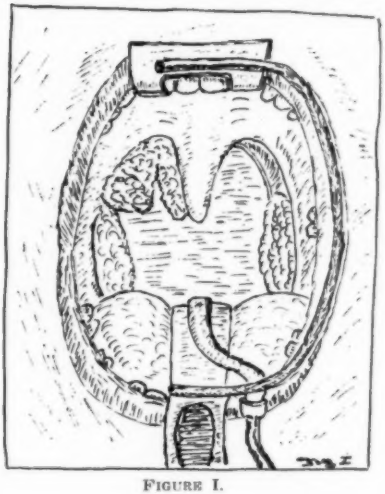

FIGURE I.

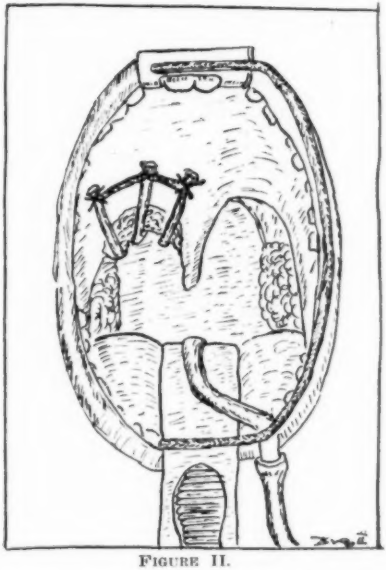

FIGURE II.

C.E.T., of Wanganui, a man aged fifty-one years, on June 21, 1930, consulted Dr. W. Macdonald, complaining of a sore throat of ten weeks' duration. The throat had not been painful, but the patient had the sensation of a lump in the back of his throat. This lump he could actually see and feel. He stated that he had had no bleeding and no bad taste, but he had been getting a lot of phlegm in his mouth. Examination revealed a raspberry-like lobulated mass growing from and involving the soft palate near its free margin, and extending from the apex of the tonsil to and behind the uvula. This mass was red, with tiny lobulations, and in part covered by an ivory to grey membrane (see Figure I). The edge was hard and indurated.

I was asked to see him with a view of treating him with radium. His teeth were very defective and much pyorrhœa was present. On June 23, 1930, his teeth were extracted and a rigid mouth toilet adopted. On June 29, 1930, under

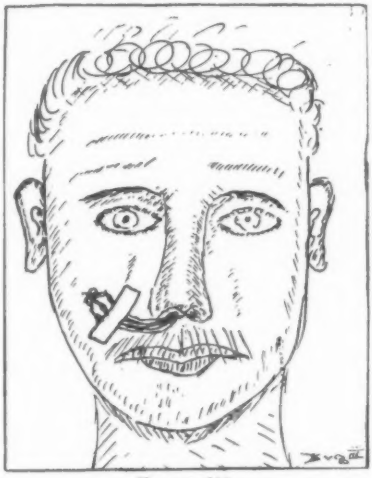

FIGURE III.

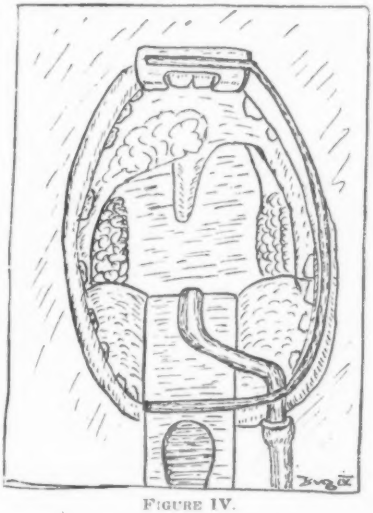

FIGURE IV.

chloroform anæsthesia, a piece having been removed for biopsy, the whole growth was subjected to "slow cooking" with the diathermy until the whole mass was blanched.

Then eight radium needles, each containing 0·6 of a milligramme of radium of one centimetre active length and 0·6 millimetre platinum screenage, were introduced about one centimetre apart. They were introduced from before backwards and from back forwards and as far as possible stitched in. There was some difficulty in getting healthy tissue to stitch into. Linen threads from the needles were brought out of the mouth within rubber tubing and affixed to the cheek with leucoplast.

Owing to the almost perpetual motion of the soft palate, these rapidly worked loose. He shed one needle at 4 a.m. the following morning, two needles at 10 a.m. the following morning, three needles by 11 a.m. on the second day, one needle on the third day, and one needle on the fourth day.

At this operation the posterior portion of the ascending ramus of the mandible on the affected side was removed to give better access to superficial radium later.

Realizing the inadequacy of the dose, we considered driving the needles in from the exterior, which did not promise an ideal access. Dr. Rhind suggested using an intranasal rubber tube.

On July 11, 1930, we loaded three pieces of small calibre rubber tubing with two threaded needles apiece (see Figure V). Here we again used 0·6 milligramme of radium to one cubic centimetre of linear intensity, but with only 0·5 millimetre of platinum screen.

These needles were placed in tubes, as in Figure V, about one centimetre separating the two needles in each tube. A silk tie was tied tightly above and below each needle. Next the tubes were wired together one centimetre apart, as shown in Figure V, with rubber tubed covered wire. Next two silk stitches were introduced through the soft palate, one in the middle line and anterior to the base of the uvula and about one centimetre forward of the free margin, the other stitch about the same transverse level, but lateral and anterior to the pillars of the fauces. The stitches were introduced in such a manner that we had two loops on the nasal side of the palate and two pairs of free ends on the buccal side of the palate. These loops were hooked forward into the mouth, and the free ends of two of the three radium loaded tubes were threaded through the lateral loop, whilst the free end of the third tube was threaded through the medial loop.

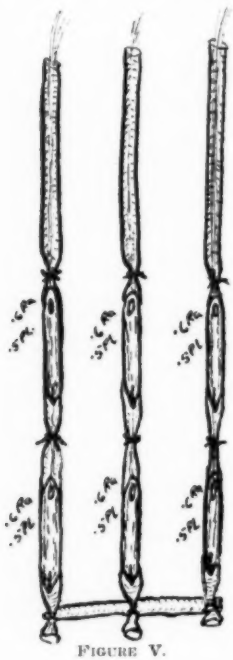

FIGURE V.

A rubber catheter threaded with a silk thread was introduced through the nose, and one end of the catheter and thread brought forward into the buccal cavity, and to this thread were tied the three free ends of the rubber tubes. In this manner by removing the catheter the free ends of the tubing presented through the anterior nares (see Figure III).

By now threading the two outside corners of the tethered ends of the rubber tubes between the pairs of buccal stitches and drawing the loops and tubing taut, the radium needles came to be three upon the nasal side of the soft palate and three on the buccal side. The stitches were tied to hold them in this situation (see Figure II). So they remained unmovable and the patient was moderately comfortable for ten days. During the last five days he got a slight burning sensation on the back of the tongue, which did not last long and was not severe enough to produce a pellicle on his tongue.

In this manner he had 864 milligramme hours distributed over ten days. Daily a few drops of 10% argyrol were introduced through his nose as a prophylactic measure.

On July 3, 1930, some ten days after their removal, as I have endeavoured to depict in Figure IV, there was a flattened congested area covered with the typical radium pellicle, with the same inflammatory reaction around, and I believed his palate was going on satisfactorily.

I determined to leave him for five weeks and then irradiate his neck with a Columbia paste collar.

There had been no palpable glands. I might mention that the pathologist reported: "Specimen is an actively growing squamous cell carcinoma (epithelioma). There are many cell nests even in the deeper parts."

On September 7, 1930, examination revealed a very healthy looking palate. The soft palate was slightly elevated on what had been the affected side. A Columbia paste collar was made, it being fifteen millimetres thick. This collar was loaded with forty-one milligrammes of radium in such a manner that the lower half of his parotid gland, submaxillary triangle, carotid triangle and sterno-mastoid

muscle region were subjected to superficial radiation with radium for ten days continuously. He then had a rest from the collar for nine days, when a fresh collar was applied radiating the same area with forty-four milligrammes of radium for another ten days.

Thus his glandular area was subjected to 20,400 milligramme hours of radium. This exposure to radium which was in the form of 1·0 milligramme, 1·3 milligramme and 2·0 milligramme needles screened with 0·8 millimetre of platinum, produced a severe skin reaction of such severity that he had a weeping eczema and peeled over his glandular distribution. This was not painful and caused him no serious inconvenience a week after the removal of his collar.

I believe this will prove a most satisfactory and easy mode of applying radium to the soft palate when there is so much destruction that one cannot get healthy tissue into which the needles can be stitched. It is very much safer than tying them the one to the other when introduced in pairs point to point.

With radon one can implant seeds and cut the threads short and hunt for the seeds in the hay stack after they are spent. I prefer the above described method.

CARCINOMA OF THE JEJUNUM.

By ALAN NEWTON,

Honorary Surgeon, Melbourne Hospital, Melbourne.

THIS case is of interest for the following reasons: (i) Carcinoma of the jejunum is comparatively rare. (ii) A correct pre-operative diagnosis was made. (iii) Despite the advanced age of the patient, resection of the growth was followed by an uneventful recovery.

The patient, a male, aged seventy-five years, was seen in February, 1931, in consultation with Dr. W. K. Davenport. He stated that he had been quite well until September, 1930, when he suddenly experienced an attack of pain in the right hypochondrium which "doubled him up." Shortly after the attack began, he vomited a large quantity of bile, after which the pain gradually subsided and he felt quite well on the following day. In October, 1930, he had a precisely similar attack. In November he was free from discomfort, but in December suffered from a third attack similar in character to the other two. At this time it was thought that the pain might be due to biliary colic and a cholecystographic examination was made. The dye was administered by the mouth and the gall bladder was not visualized. During January, 1931, attacks of pain occurred every third day. The pain in these attacks in January was now felt in the region of the umbilicus. It was accompanied by a feeling of upper abdominal distention and was followed by profuse vomiting of bile stained fluid. On one occasion the vomited material filled two bedroom basins. There was no alteration in the habitual action of the bowel, and the motions contained no blood or mucus. He had had no past illnesses of any importance. Except that it was obvious that the patient had lost weight, routine examination revealed nothing abnormal. I did not see him during an attack, but was informed that upper abdominal distention was then easily visible, though no reverse peristaltic waves had been observed. No abdominal tumour was palpable, nor were there any areas of rigidity or tenderness. As the symptoms clearly indicated a condition of steadily increasing obstruction in the upper part of the alimentary tract, and as the fact that the vomit contained bile proved that this obstruction was below the middle of the second part of the duodenum, a provisional diagnosis of carcinoma of the jejunum was made. A barium meal examination showed that the stomach was almost completely empty in six hours, and that it was normal in shape, size, tone and position. The duodenal cap was regular. The second and third parts of the duodenum were moderately dilated, and the first six inches of the jejunum were enormously dilated. A large amount of barium was found in this coil twelve

hours after the administration of the meal, and active peristalsis was observed. The patient had been given a double meal and some of the barium taken twenty-four hours before was in the colon. Some extra-luminal shadows, suggesting diverticulosis of the sigmoid colon, were seen. The jejunal obstruction was in the vicinity of these shadows (see Figure I), but, owing to the absence of symptoms and signs of diverticulosis, it was not thought that the obstruction was due to an inflammatory adhesion, and the diagnosis, carcinoma of the jejunum, was confirmed.

FIGURE I. Dilated jejunal coil proximal to the carcinoma and diverticulosis of the sigmoid colon.

Two days were spent in preparing the patient for operation. In order to increase the glycogen storage in the liver, large quantities of lactose were added to all fluids administered. Lactose is much less nauseating than glucose and, therefore, can be given in larger amounts. Nutrient enemata of 5% glucose in 300 cubic centimetres (ten ounces) of normal saline solution were given every four hours. It was not thought necessary further to increase the chloride content of the blood by giving saline solution intravenously.

The anæsthetic selected was ethylene and oxygen, as there is no doubt that this is incomparably the best anæsthetic for upper abdominal operations on elderly patients. The abdomen was opened by a right epigastric incision reflecting

the rectus muscle outwards. A carcinoma of the scirrhous type was found fifteen centimetres (six inches) distal to the duodeno-jejunal flexure. No enlarged glands were seen and a careful search of the abdominal cavity failed to reveal any secondary growths. Examination of the gall bladder revealed nothing abnormal. The small bowel was resected from a point ten centimetres (four inches) above the growth to a point fifteen centimetres (six inches) below it and an end-to-end anatomosis performed with suture of number one chromicized catgut, so placed

FIGURE II. External appearance the growth.

FIGURE III. Internal appearance of the growth.

that the dilated upper end was pleated into the contracted lower end. The growth was a small annular carcinoma which completely surrounded the lumen of the bowel and had infiltrated deeply into the muscle and subserous coats. The peritoneum over the growth was puckered and wrinkled. Obstruction was almost complete, as was shown by the fact that when the bowel, above the constriction, was filled with water, only a very fine trickle passed through. The mucous membrane surface of the growth showed some superficial ulceration (see Figures II and III). Microscopical examination revealed an active adenocarcinoma with wide infiltration of the muscle coat. There was no colloid change (see Figure IV). Six hours after the operation the patient was allowed to drink water freely. Two teaspoonfuls

of lactose were dissolved in each glass of water. He was given "Benger's Food" with lactose on the day after operation and thenceforward his diet was steadily increased. Purgatives were not administered during the first week, a bowel action being obtained every second day by an enema. He made an uninterrupted recovery

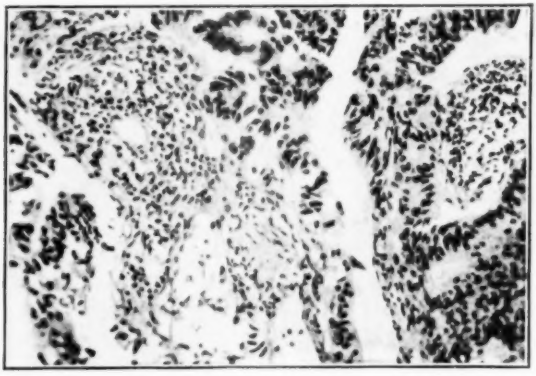

FIGURE IV. Microscopical appearance showing infiltration of the muscle coat. × 170.

and was discharged from hospital in three weeks. When seen two months after operation, he stated that he felt very well and had gained 12·6 kilograms (twenty-eight pounds) in weight.

Acknowledgements.

I am indebted to Dr. H. M. Hewlett for the X ray examination of this patient and to Dr. R. Wright-Smith for the pathological report.

FIBROMA OF TENDON SHEATH.

By R. J. WRIGHT-SMITH.

[From the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne.]

TUMOURS of tendon sheaths are extremely rare. Fibromata are amongst the more uncommon and in the literature at my disposal I have only been able to find fourteen of these. The following case, therefore, seems worthy of record.

T.L., aged twenty-one years, was admitted to the Melbourne Hospital on March 4, 1931, with contraction of the ring finger of the left hand. While he was playing football six months previously, this finger had been caught in an opponent's sweater. It shortly became swollen and discoloured, with limitation of movement. Prior to this injury he had not noticed any abnormality. The deformity did not progress in the ensuing period.

The left hand showed a flexion contracture of the ring finger when the other fingers were extended, and a hard subcutaneous cord was palpable in the palm in the line of the tendon to the ring finger. The condition resembled so closely an early or middle stage Dupuytren's contracture of the ring finger that in spite of the age of the patient, this pre-operative diagnosis was made. The voluntary

movement of the ring finger was limited. There was full flexion of the metacarpo-phalangeal joint, but extension of all joints was limited.

At operation the bag of a sphygmomanometer was used as a tourniquet, and an incision was made in the line of the flexor tendons of the ring finger. As soon as the fibres of the palmar fascia were divided, a cylindrical shaped, white, encapsulated and freely mobile tumour presented. This was readily dissected free. It extended proximally into the hand from about the level of the distal transverse crease of the palm. In this region it was attached by a pedicle about one-half the thickness of the tumour to the profundus tendon of the ring finger, at the entry of the tendons into the tendon sheath. The tumour appeared to be growing from the tendon and not from the sheath. The tumour was excised and the wound closed.

FIGURE I.

The tumour (see Figure I) is roughly cylindrical in shape and is three centimetres in length and approximately half a centimetre in diameter. The surface is smooth and a definite capsule appears to be present. A small portion of tendon is firmly united to its lower pole. The tumour is very tough in consistency, and has a definitely fasciculated appearance in longitudinal section. It contains very little blood and is uniformly white in colour.

Microscopical examination (see Figures II and III) reveals very few cells embedded in an abundant matrix, which has undergone hyaline transformation. Blood vessels are extremely scanty. At the periphery of the tumour, cells are more numerous, but show no great activity.

FIGURE II.

FIGURE III.

Acknowledgements.

I am greatly indebted to Mr. W. Allan Hailes for the clinical notes, and to Mr. E. S. J. King for the bibliography.

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F. Torek: "Fibroma of the Sheath of a Tendon," *New York Medical Journal*, 1905, Volume LXXXII, page 20.

Reviews.

CLINICAL DIAGNOSIS.

"CLINICAL EXAMINATION AND SURGICAL DIAGNOSIS"[1] is an English edition of the work by Professor Lejars, of Paris, of which two French editions have appeared. The translator is Dr. Helen C. Scott, who forewarns us that she has tried to preserve a great deal of the French spirit of the original. This she could hardly avoid, since, though the delightful staccato intimacy of the opening chapters diminishes as the writer warms to his subject, the graphic Gallic garrulity is sustained throughout. The book is divided into thirteen chapters anatomically arranged, the injuries and diseases to be met with in a given region are considered and the method of examination to be followed is explained in great detail and profusely illustrated. The methods of examination are strictly clinical, except for occasional arbitration or confirmation by radiograph. The illustrations consist of a few diagrams, some anatomical drawings and a very large number of photographs. All the figures illustrating clinical examination have been taken from patients actually suffering from the condition under consideration, and have been collected by the author over a period of about forty years. They form a unique collection, and fully serve their purpose, though it may be admitted that their reproduction leaves something to be desired. The diagnostic argument is frequently reinforced by case histories from the author's experience, and these are always interesting.

The outstanding feature of the book is the clear and exhaustive description of the methods of palpation. There is reason to admit that of all methods of examination, palpation as performed by the average student and practitioner, save in certain special spheres as gynæcology and midwifery, is the most haphazard and, comparatively speaking, the least fruitful, yet the expert in the art is popularly rated far above his fellows who lack the flair. It may be that the method being instinctive and impulsive and the too eager student finding himself restrained by his teacher until he has fully exercised his powers of observation, there tends to be created a subconscious suggestion that to spend much time on palpation is a sign of clinical inefficiency. Moreover, either because of the complex created or because his futile fumblings lead him to negative or erroneous conclusions, he

[1] "Clinical Examination and Surgical Diagnosis," by Félix Lejars; translated by Helen C. Scott, M.R.C.S., F.R.C.P.; 1931. London: Jonathan Cape. Royal 8vo., pp. 872, with illustrations.

comes to distrust his fingers and to turn to X ray examination or to exploratory operation to enable him to arrive at a decision. Dr. Lejars, too, gives the emphatic advice, "Look before touching," but while he impresses the importance of informed observation, he devotes more space to the more difficult method of palpation: of the 1,099 illustrations, more than one-third are descriptive of how to arrive at clinical conclusions through the fingers. Percussion is, of course, merely a refinement of palpation.

Dr. Lejars's previous work, "Urgent Surgery," is a classic. This book is likely to be more widely read, as it will appeal to an even larger number of the profession. The best informed may learn from it, and the humblest of the rank and file will not complain that it is "technical."

PATHOLOGY FOR NURSES.

THE time has long passed when nurses were automatons, assistants who carried out instructions without having a proper understanding of what they were doing or why they were doing it. Nurses are given regular courses of instruction and are made to pass searching examinations. Part of their instruction must be in the realm of pathology and bacteriology. As a guide to nurses so engaged, Dr. C. G. Sinclair has written "Microbiology and Elementary Pathology."[1] He has done his work well. The book is divided into three parts: Part I on microbiology, Part II on laboratory exercises in microbiology and Part III on elementary pathology. The information given in the several sections is full enough without being too full; it will allow any nurse to take an intelligent interest in her work and to cooperate when necessary. To many chapters the author adds a final paragraph setting out the importance for the nurse in her work of the contents of the chapter. Some of the information would be useful to a nurse desirous of training as a technical assistant in a clinical laboratory. The author suggests in his preface that the book may prove useful to those instructing nurses, in order that the progressive orderly sequence of the lessons may be preserved. We are inclined to agree with him. This book may be confidently recommended.

EMBRYOLOGY.

PROFESSOR C. S. SIMPKINS, of the University of Tennessee Medical School, has written a textbook on human embryology.[2] He intends it for the use of students interested primarily in the development of the human body. He has, where possible, emphasized the functional aspect of development, and this, in our opinion, must be commended.

The author starts with a short chapter on the reproductive organs of the adult, and then includes one on cyclical phenomena in the female. The introduction of these chapters is not really necessary, though their inclusion may make for completeness. Hereafter the book is planned on conventional lines. There are chapters devoted to maturation of germ cells, cleavage and implantation, development of external form, development of face and jaws, the fœtal membranes. Subsequent chapters deal with the several systems. The chapter on the development of the vascular system is well planned. The author discusses both the monophyletic and polyphyletic theories of the development of the blood. He does not express his own views on the question, but from his writing it would appear

[1] "Microbiology and Elementary Pathology for the Use of Nurses," by Charles G. Sinclair, B.S., M.D.; 1931. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 362, with illustrations, some in colour.

[2] "Textbook of Human Embryology," by C. S. Simkins, Ph.D.; 1931. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 469, with 263 illustrations, some in colour.

that he favours the former. The chapter on the development of the digestive system and that on the urogenital system are good. The rectum receives rather scant attention, being referred to in both these chapters. In our opinion, more attention might have been paid to abnormalities of the hinder end of the gut. The chapter on the glands of internal secretion is rather scrappy.

Taken as a whole, the book is likely to be useful. It is a little uneven perhaps, but it has the advantage of being well illustrated; the line drawings in particular are valuable and serve well for teaching purposes. It is a pity that the author's bibliography is almost entirely American. The publishers deserve praise for the general make-up and printing. Both are excellent.

HANDBOOKS ON HISTORY.

A PROPER appreciation of medicine as an art or a science cannot be obtained without some knowledge of its history. Hitherto the acquisition of even a smattering of knowledge of medical history has entailed a vast amount of reading. The need has long been felt for some handy volume setting out the salient points and summarizing the historical progress of medicine. An endeavour is made to supply this want in a series of volumes entitled "Clio Medica."[1] The editor, E. B. Krumbhaar, mentions in his preface that hitherto works on this subject have been either too ponderous or too skimpy. He states that "each volume will aim to present the story of some individualised phase of the history of medicine in such compact, connected, convincing and reasonably complete form that the medical undergraduate, the specialist, the busy general practitioner and the 'intelligent layman' will be attracted to a few hours' reading. . . ." Five volumes have so far been published, and they are sufficiently attractive as to appear likely to fulfil their editor's hopes.

In Volume I, "The Beginnings, Egypt and Assyria," Warren R. Dawson discusses the fascinating story of ancient Assyrian and Egyptian medicine and magic. Volume II, "Medicine in the British Isles," is written by D'Arcy Power, who deals ably with this most important chapter in the history of medicine. In Volume III George W. Corner skilfully unfolds the story of the growth of knowledge of human anatomy. Volume IV, "Internal Medicine," is the work of Humphrey Rolleston; it contains a brief history of the various schools of medicine, and shows how the science of internal medicine has grown. John F. Fulton is the author of Volume V, "Physiology"; this is the largest of the five volumes and perhaps the most interesting.

The writing is all good, some of it quite charming. Each volume is small—the largest contains but 140 pages—and may be carried comfortably in the coat pocket. The subjects are necessarily dealt with briefly, but nowhere is the brevity such as to make the reading dull; rather does the reader wish there were more of it. Every medical practitioner should have a copy of each of these five delightful little books in his possession.

MALIGNANT DISEASE.

DR. WILLY MEYER, Emeritus Professor of Surgery of the New York Postgraduate Medical School, has written a book entitled "Cancer: Its Origin, Its

[1] "Clio Medica: A Series of Primers on the History of Medicine," edited by E. B. Krumbhaar, M.D. I—The Beginnings: Egypt and Assyria, by W. R. Dawson, F.R.S.E.; crown 8vo., pp. 95; price, $1.50 net. II—Medicine in the British Isles, by Sir D'Arcy Power, K.B.E., F.R.C.S.E.; crown 8vo., pp. 99; price, $1.50 net. III—Anatomy, by G. W. Corner, M.D.; crown 8vo., pp. 99, with illustrations; price, $1.50 net. IV—Internal Medicine, by Sir H. Rolleston, Bart.; crown 8vo., pp. 101; price, $1.50 net. V—Physiology, by J. F. Fulton, M.D.; 1930. New York: Paul B. Hoeber. Crown 8vo., pp. 156, with illustrations. Price: $1.50 net.

Development and Its Self-Perpetuation."[1] This is a peculiar book; we have not seen anything quite like it. The author has built up an elaborate hypothesis and has deduced from it what he describes as "THE Therapy" [*sic*] in inoperable cancer. He has written this book from the point of view of the student who starts out to fathom the cancer problem with the help of the literature. Such a writer finds himself, he points out, thrown upon his own resources, upon speculation, however much this may go against the grain. He does not describe facts from which to draw conclusions, but draws the essence from the conclusions of others to get at the facts; he does not use results to arrive at deductions, but starts at deductions to obtain results. This method involves, so Dr. Meyer states, the intense application of the personal equation. His book "does not lend itself to fact-finding," but appears "well adapted to path-finding." He offers his book as an experiment in path-finding. Were this book to be judged from the point of view of hard fact, it would be counted as of little worth. An author's objective must always be considered. If a writer sets out to tell a fairy tale, he must not be pilloried with the cold-blooded historian. Dr. Meyer has made intense application of the personal equation, he has drawn freely from the conclusions of others and he has endeavoured to obtain results by this means. The book is not all fairy tale. Even if it were, fairy tales are often attractive.

Cancer, to the author, is an ordinary lawful reaction of the tissue to certain systemic and local morbid conditions of the human organism. Cancer should not be considered as a local tumour formation, without regard being paid to changes occurring at the same time in the general system. Cancer is a systemic disease and is the effect of two simultaneously active chronic irritations, the one systemic and the other of local character. Neither the systemic nor the local chronic irritation is specific for cancer; both these types of irritation are harmless as far as the production of cancer is concerned, when they are not acting in conjunction. The systemic chronic irritation has its origin in irregularities of the nervous system and in disturbances of the physico-chemical condition of the body fluids, either of which may precede the other. The local chronic irritation has its origin in an abnormal behaviour of the tissue cells. Both forms of irritation are discussed in considerable detail. Cancer susceptibility, it is argued, can be either inherited or acquired.

The author holds that "it can be said with a considerable degree of definiteness, that alkalosis of the blood predisposes to cancer and that this alkalosis . . . . is not the consequence of, but precedes tumor formation." What he regards as "THE" treatment for cancer are substances capable of producing acidosis of the serum. If there were a method which could be described as "THE" treatment, it would certainly be in common use and would be producing cures. The methods for changing the alkalosis of the serum to an acidosis are: the production of fever, either infectious or aseptic; the administration or the mobilization in the system of the calcium ions; the administration of acids and the injection of acid colours.

Dr. Meyer's book might be used as the basis for a hundred arguments. It certainly shows the trend of much present-day cancer investigation. If he does nothing else than make his readers think, he will have gone a long way towards achieving his purpose.

URGENT SURGERY.

"WHILE writing the present volume I have pictured a patient stricken with an acute abdominal catastrophe and a comparatively isolated surgeon called upon to carry out appropriate treatment. Should these pages help the latter to save

[1] "Cancer: Its Origin, Its Development and Its Self-Perpetuation: The Therapy of Operable and Inoperable Cancer in the Light of a Systemic Conception of Malignancy: A Research," by W. Meyer, M.D.; 1931. New York: Paul B. Hoeber. Royal 8vo., pp. 460, with portraits. Price: $7.50 net.

the former, their main object will be fulfilled." These are the words with which Hamilton Bailey opens the preface of his most acceptable book, "Emergency Surgery."[1] There is scope for a book such as this, particularly in Australia and New Zealand where many surgeons may be described as "comparatively isolated." The present volume is the first of a series of two. It is devoted to the abdomen and pelvis. In the second volume the author will deal with the surgical emergencies of the head, neck, spine, thorax and the extremities.

The chapters are arranged according to important lesions and various regions. Thus there is a chapter on peritonitis and one on acute appendicitis. In the latter the author lays stress on the value of the Ochsner-Sherren treatment, and of the incision of a pelvic abscess through the rectum. The section on perforation of gastric or duodenal abscess is clearly written, and may be read with profit by any surgeon. The statement that "rectal injection as a method of treatment of acute intussusception is so uncertain that it is now rightly obsolete," will not be accepted by all Australian surgeons. The success obtained by Hipsley with rectal injection at the Royal Alexandra Hospital for Children, Sydney, which was reported in *The Medical Journal of Australia*, and the definite guidance given by him for the use of this method, belie the author's statement. We are glad to note the author's recommendation of posterior colpotomy for the draining of a pelvic abscess—a procedure too seldom adopted. It would have been more satisfactory had he discussed in detail the indications for its use.

The few criticisms offered do not denote any antagonism to this book. On the contrary, we are enthusiastic as to its value. It would be impossible for any surgeon to produce a book on emergency surgery that would satisfy everyone. We would that the author did not descend in places to the use of clinical jargon, as he does on page 80, in describing the after-treatment of peptic ulcer. The book is sound and will be welcomed both by the "isolated" surgeon and his more fortunate colleague.

JOINT INJURY.

"INJURIES TO JOINTS," by Sir Robert Jones, has reached its third edition.[2] This little book is one of the "Oxford Medical Publications." It was first published in 1915 as a guide to war surgeons. That it will be of use to surgeons in civil practice and general practitioners is undoubted. The book is so well known that it is unnecessary to do more than announce the appearance of this edition.

THE VETERINARY JOURNAL.

THE March, 1931, number of *The Veterinary Journal* has been received. This is a special surgical number. The articles are devoted to a multiplicity of subjects, such as bowel resection and lateral anastomosis in the dog by J. G. Wright, oophorectomy in the mare by J. G. Wright and R. Machin, the ventricle stripping operation for roaring by F. T. E. Hobday, treatment of fracture by wiring by J. McCunn and C. Formston, and radiological problems in animals by R. W. A. Salmond. The issue is of unusual interest.

[1] "Emergency Surgery," by Hamilton Bailey, F.R.C.S. (England); 1930. Bristol: John Wright and Sons Limited. Volume I. Royal 8vo., pp. 380, with illustrations.

[2] "Injuries to Joints," by Sir Robert Jones, Bart., K.B.E., C.B., Ch.M. (Liverpool), F.R.C.S. (England, Ireland and Edinburgh), F.A.C.S. (U.S.A.); Third Edition; 1930. Oxford University Press (Medical Publications). Foolscap 8vo., pp. 195, with illustrations.